4th Edition

Clinical Pearls *in* GENERAL MEDICINE

FCPS-Part II & IMM

(Examination schemes for short and long cases)

Notes for Short & Long Cases

(Fellowship Exam in Internal Medicine)

Salient Features

- Clinical schemes for short cases & Long cases
- Important points for history taking
- Explaination of examination findings
- Causes of common clinical signs
- Important viva question (new edition)

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Chapter – 1

Short Case Examination in Nervous System

1.1. Scheme for Examination of Motor System of Lower Limb

Command: Examine Motor System of Lower limb & Do Relevant

- Make a deep observation while approaching patients for **Foleys catheter, Deformities, central venous catheter and walking aids.**
- Approach from **right side**
- Introduce yourself
- Ask patient name & assess speech (Dysarthria, motor/sensory dysphasia).
- Take Consent for examination.
- Make appropriate Position for examination (Supine)
- Exposure (ideally ask patient to uncover his limbs to get an idea about power in upper limb).
- Inspect bilaterally for

Hair loss (may indicate neuropathy or ischemia)

↓

Fasciculation (Spontaneous and Induced, at four areas Bilateral thighs and lower legs)

↓

Bulk (check with measuring tape and compare bilaterally if visible wasting present)

After that check for

Tone and compare it bilaterally around both ankles, knees & hip joints while fixing proximal and distal joints

↓

Power bilaterally in all major muscles group (check against resistance if power is more than grade 3)

↓

Reflexes- compare bilaterally in ankle and knee joints (Check for ankle and patellar clonus if reflexes are brisk)

↓

Planter response bilaterally

↓

Heal shin Test if power is greater than grade 3 (for cerebellar sign assessment)

Scheme for Relevant examination

If on examination of lower limbs power is reduced with hypertonia & hyper-reflexia & extensor planter response on one side (hemiparesis) then:

Proceed quickly to **upper limb examination**

↓

Assess quickly -- **tone, power, reflexes, and finger nose test** (in upper limbs bilaterally)

↓

Move to cranial nerves examination (quick assessment)

- **Show your teeth** (for functional impairment in 7th cranial nerve)
- **Close your eyes don't let me open** (for functional impairment in 7th cranial nerve)
- **Extrao-cular movements** (for functional impairment in 3rd, 4th and 6th cranial nerves)
- **Open mouth, say Aaaah and check position of uvla** (for functional impairment in 9th and 10th cranial nerves)
- **Cough** (for functional impairment in 9th and 10th cranial nerves)
- **Protrude tongue** (for functional impairment in 12th cranial nerve)

↓

Examine **Sensation** to

- Pin prick sensation bilateraly in upper/lower limb
- Cortical sensation
    - Two point discrimination
    - Localization,
    - Sensory inattention in suspected cerebral stroke patients

↓

- **Gait** if power is greater than grade 3 – Romberg test and heel to toe

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Move to examination to find out possible cause & complication:

Pulse (irregular- atrial fibrillation)

↓

Blood pressure (Hypertension)

↓

Auscultate Carotid arteries bilaterally for Bruit (carotid atherosclerosis)

↓

Xanthelasmas (dyslipidemia)

↓

Precordial auscultation (valvular lesion leading to emboli)

↓

Back of Chest for Aspiration pneumonia (stroke complication)

↓

Bed sores (stroke complication)

↓

1.2: Scheme for spinal cord Compression

If on examination of lower limb findings are suggestive of spastic parapareses (upper motor neuron signs in both lower limbs then proceed to following examination

↓

Examine **Pin Prick** sensation and **Position Sense**

↓

If impaired find out level ⇨

If at T4 --- Examine sensation in upper limb, if impaired try to find out level in upper limb and check motor system too

↓

If level below T4

↓

- **Finger nose test** (to check for cerebellar signs in case of multiple sclerosis—two space lesion)
- **Extra-ocular movement and Nystegmus** (to check for intraneuclear ophthalmoplegia to rule out multiple sclerosis)

↓

Examination of Back for

- ✧ **Spinal deformity** (Gibbus formation in spinal Tuberculosis)
- ✧ **Spinal Tenderness** (metastatic disease) and
- ✧ **Bed sores** (complication of parapresis)

↓

Chest auscultation at apices for pulmonary tuberculosis

↓

Lymph nodes for Tuberculosis/lymphoma.

↓

Offer **fundoscopy** (Multiple sclerosis).

↓

Gait if power is greater than grade 3.

1.3: Scheme for spastic paraparesis with impaired position sense

If on examination of lower limbs finding are suggestive for Spastic paraparesis with ***intact pin prick & impaired position sense***

↓

Check for

- Past pointing (for cerebellar disorder)
- Extra-ocular movements & Nystegmus

↓	↓	↓	↓
Look for: Pescavus High arch palate Precordium (HOCM) Kyphosis Fundus	**Look for:** **Paler** - B12 deficiency anemia **Glossitis** Precordium (**Flow murmur** due to anemia)	Look for: Argyll Robertson pupil Precordium (AR)	Examine Fundus- Optic neuritis
Fredrick's ataxia	**Subacute combined degeneration**	**Tabesdorsalis**	**Multiple sclerosis**

1.4: Scheme for spastic paraparesis with no sensory impairment

If Lower limbs examination findings are suggestive for **Spastic parapresis** with **No Sensory level or sensory impairment -**

↓

- ➤ Quickly proceed to upper limb (to check involvement of upper limbs) ,
- ➤ Quick assessment for bilateral
  - o Bulk / Fasciculation
  - o Tone
  - o Power
  - o Reflexes

↓

- ➤ Check for **Past pointing** by finger nose test bilateraly (for cerebellar signs - possible multiple sclerosis)
- ➤ Examine **Cranial nerves**

(Quickly assess following ----

↓

- **Show your teeth** (for functional impairment in 7th cranial nerve)
- **Close your eyes don't let me open** (for functional impairment in 7th cranial nerve)
- **Extrao-cular movements** (for functional impairment in 3rd, 4th and 6th cranial nerves)
- **Open mouth, say Aaaah and check position of uvla** (for functional impairment in 9th and 10th cranial nerves)
- **Cough** (for functional impairment in 9th and 10th cranial nerves)
- **Protrude tongue** (for functional impairment in 12th cranial nerve)

↓

- ➤ Examine Jaw Jerk (for motor neuron disease)

↓

- ➤ Examine Cortical Sensation (two point discrimination, localization, sensory inattention for bilateral cortical stroke/tumor i.e parasagittal meningioma)

↓

- ➤ Fundus examination (Multiple sclerosis)

↓

- ➤ Gait if power is greater than grade 3.

Differential diagnosis for spastic paraparesis without sensory level:

- Motor neuron disease
- Parasagittalmeningioma
- Bilateral anterior Artery circulation stroke.
- Hereditary Spastic paraparesis
- Multiple sclerosis

1.5: Scheme for flaccid paraparesis

If you found Flaccid Paraplegia/parapresis on examination of lower limbs

With or without sensory loss in gloves & stocking distribution

↓

- ➢ Quickly proceed to upper limb (to check involvement of upper limbs)
- ➢ Quick assessment for bilateral
    - o Bulk / Fasciculation
    - o Tone
    - o Power
    - o Reflexes
    - o Sensory system examination of upper limb (pin prick sensation)

↓

- ➢ Quickly assess cranial nerves
    - **Show your teeth** (for functional impairment in 7th cranial nerve)
    - **Close your eyes don't let me open** (for functional impairment in 7th cranial nerve)
    - **Extrao-cular movements** (for functional impairment in 3rd, 4th and 6th cranial nerves)
    - **Open mouth, say Aaaah and check position of uvla** (for functional impairment in 9th and 10th cranial nerves)
    - **Cough** (for functional impairment in 9th and 10th cranial nerves)
    - **Protrude tongue** (for functional impairment in 12th cranial nerve)

↓

- ➢ Then do rapid screening for the possible **cause**
    - Pulse
        - o **resting tachycardia** for autonomic neuropathy in Guillain-Barré Syndrome- GBS), and

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    - **arrhythmias** for autonomic neuropathy in Guillain-Barré Syndrome-GBS)
- Blood pressure
    - Hypertension
    - Postural drop at the end of examination- for autonomic neuropathy in GBS)
- Thyroid enlargement by deglutination (for periodic paralysis)
- Chest expansion with measuring tape (for respiratory muscle involvement)
- Bed sores (complication of paraplegia)
- Blue line on gum (lead poisoning)
- Finger prick mark (Diabetes Mellitus).

Differential diagnosis

- Guillain-Barré SyndromeGBS.
- Chronic inflammatory demyelinating polyneuropathy (CIDP)
- Periodic paralysis
- Lead poisoning
- Botulism
- Diabetes mellitus

1.6: Scheme for proximal muscle weakness

If on examination of lower limbs you have found just proximal muscle weakness with no other abnormality
(Intact reflexes, no sensory loss)

↓

- Quickly proceed to upper limb.
- Quick assessment of Power in upper limbs
    - Fatigability Test (Myasthenia Gravis)
    - Muscle Tenderness (Myositis)

↓

- Move to cranial nerves examination
  - Sustained up ward gaze Test
  - Extraocular movements
  - Show teeth, smile
  - Say aaaaah, protrude tongue, cough
  - One breathe count
  - Nasal speech
  - Week Gag reflex.

↓

  - Neck lift against resistance

↓

  - Spine for kypho-scoliosis (Muscular Dystrophies)
  - Precordium for cardiomyopathy
  - Pallor
  - Thyroid enlargement
  - Skin changes for Dermatomyositis (**Gottron papule, Heliotrope rash, Shaw**
  - **sign)**
  - Cushingnoid features – Moon face, Buffalo hump, Pupurish straie,
  - Hypertension etc (itrogenic – therapeutic steroid use)

Differential diagnosis:

- Myasthenia gravis
- Myositis (Polymyosistis, Dermatomyositis)
- Muscular Dystrophies
- Cushing syndrome
- Periodic paralysis

1.7: Scheme for Examination of motor system of upper limb

Command: Examine Motor System of upper limb & do Relevant (Mostly for cerebellar lesion)

- Make a deep observation while approaching patients for **Foleys cathete** **Deformities, central venous catheter and walking aids.**

- Approach from **right side**
- Introduce yourself
- Ask patient name & assess speech (Dysarthria- Scanning speach, motor/sensory dysphasia).
- Take Consent for examination.
- Make appropriate Position for examination (Sitting preferably)
- Exposure (ideally ask patient to uncover his limbs to get an idea about power in upper limb).
- Inspect bilaterally for

Hair loss (may indicate neuropathy or ischemia)

↓

Fasciculation (Spontaneous and Induced, at four areas bilaterally

↓

Bulk (check with measuring tape and compare bilaterally if visible wasting)

- After that check for

Tone and **compare it bilaterally** around wrist, elbow & shoulder joint with fixed proximal and distal joints

↓

Power bilaterally in all major muscles group (check against resistance if power is more than grade 3)

↓

Reflexes- compare bilateraly in brachioradialis, biceps and triceps tendons

If **all intact** except for **Hypotonia** then examine:

- Past pointing
- Intentional tremor
- Dysdiadochokinesia
- Rebound Phenomenon

➢ Assess all cranial nerves quickly:

- **Show your teeth** (for functional impairment in 7th cranial nerve)

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- **Close your eyes don't let me open** (for functional impairment in 7th cranial nerve)
- **Extrao-cular movements** (for functional impairment in 3rd, 4th and 6th cranial nerves)
- **Open mouth, say Aaaah and check position of uvla** (for functional impairment in 9th and 10th cranial nerves)
- **Cough** (for functional impairment in 9th and 10th cranial nerves)
- **Protrude tongue** (for functional impairment in 12th cranial nerve)

↓

➢ Nystegmus and Saccadic eye movements

↓

➢ Assess Scanning Speech

(By asking patient to say Qus-Tun-Tunia)

↓

➢ Assess Lower limb rapidly (tone, power, reflexes)

↓

➢ Heel shin Test if power greater than 3

↓

➢ Pendular knee jerk

↓

➢ Gait if power greater than grade 3 to see:

- Broad based gait
- Tandem Gait
- Romberg sign

1.8: Scheme for ptosis

Command: Examine face & proceed

- Make a deep observation while approaching patients for **Foleys cathete Deformities, central venous catheter and walking aids.**
- Approach from **right side**
- Introduce yourself
- Ask patient name & assess speech (Dysarthria- Scanning speach, motor/senso dysphasia).

- Take Consent for examination.
- Make appropriate Position for examination (Sitting preferably)
- Look at face & observe for
  - Ptosis
  - Strabismus and Extraocular movements (3rd, 4th, 6th nerve/ophthalmoplegia)
  - Flattened Naso-labial folds (bilateral facial weekness)
  - Loss of lateral eyebrows (Hypothyroid)
  - Mask like face
- Look in oral cavity in detail (mucormycosis)
- Check Nasal & auricle orifices

If Bilateral Ptosis

↓

Check pupil (for light reflex)

↓

If Normal

↓

Examine Extra-ocular movements (if **Normal extraocular movements**)

↓

Move to cranial nerves examination

- Sustained up ward gaze Test
- Show teeth, smile
- Say aaaaah, protrude tongue, cough
- One breathe count
- Nasal speech
- Week Gag reflex

↓

Neck lift against Resistance

↓

Quickly proceed to upper limb.

Quick assessment of Power in upper limbs

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  - Fatigability Test (Myasthenia Gravis)
  - Muscle Tenderness (Myositis)

↓

- Spine for kypho-scoliosis (Muscular Dystrophies)
- Precordium for cardiomyopathy
- Pallor
- Thyroid enlargement
- Skin changes for Dermatomyositis (**Gottron papule, Heliotrope rash, Sha sign)**
- Cushingnoid features – Moon face, Buffalo hump, Pupurish straie,
- Hypertension etc (itrogenic – therapeutic steroid use)

Differential Diagnosis

- Myasthenia Gravis
- Muscular Dystrophies
- Myositis

If Unilateral Ptosis

↓

Examine Light reflex

↓

If dilated pupil

↓

Examine Extra ocular movements (3, 4, 6 cranial nerves – for ophthalmoplegia)

↓

5th Cranial Nerve examination (motor& sensory for cavernous sinus thrombosis)

↓

2nd Cranial Nerve examination (Cavernous sinus thromobosis/Orbital apex Syndrome)

- Visual acuity

- Visual Field
- Fundus examination

↓

Quick assessment of rest of cranial nerves

- **Show your teeth** (for functional impairment in 7th cranial nerve)
- **Close your eyes don't let me open** (for functional impairment in 7th cranial nerve)
- **Extrao-cular movements** (for functional impairment in 3rd, 4th and 6th cranial nerves)
- **Open mouth, say Aaaah and check position of uvla** (for functional impairment in 9th and 10th cranial nerves)
- **Cough** (for functional impairment in 9th and 10th cranial nerves)
- **Protrude tongue** (for functional impairment in 12th cranial nerve)

↓

Examine Neck stiffness (Post. Communicating artery aneurysm/SAH)

↓

Finger prick marks (Diabetes Mellitus)

Oral cavity/nasal cavity (Mucormycosis/Wegners disease)

Thyroid examination (Periodic paralysis/Hypothyroid ophthalmopathy)

↓

Lower limb motor and Sensory examination quickly along with assessment of planter response (Mononeuritis Multiplex)

Differential diagnosis:

- Cavernous sinus Thrombosis
- Orbital apex Syndrome
- Posterior communicating artery aneurysm
- Diabetes mellitus
- Weber syndrome
- Mononeuritis multiplex.

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Unilateral ptosis with constricted pupil

↓

Enaphthalmos (Hornors syndrome)

↓

Extra-ocular movements and Nystegmus (PICA syndrome/ Multiple sclerosis)

↓

5th cranial Nerve examination Sensory & Motor

↓

9th & 10th cranial nerve examination by Gag reflex

↓

Rest of cranial nerve assessment quickly

↓

Neck –

- Thyroid Scar (thyroidectomy)
- Palpate bilateral carotids one by one for aneurysm
- Examie cervical Lymph nodes bilateraly

↓

Auscultate lung apices (Pancost tumor)

↓

Past pointing in upper limb (Bilaterally for celebeller signs)

↓

Lower limb – Rapid assessment of motor and Sensory system along with Plante response (PICA syndrome/Syringomyelia/Multiple sclerosis)

Differential diagnosis:

- PICA syndrome
- Syringomyelia
- Multiple sclerosis
- Carotid artery aneurysm/Dissection
- Pancoast tumor
- Trauma to neck
- Thyroidectomy

1.9: Scheme for Parkinson's disease

Parkinson's disease:

- Mask like expressionless face, less blinking, staring look
- Tibutations of head
- Blephroclonus
- Drooling of saliva
- Speech – slow, tremulous, monotonous, repetition of end word
- Tremors- resting, pin rolling
- Give command to fasten/open button, count on finger to check bradykinesia
- Rigidity- increased by voluntary movement on other limb
- Check writing – micrographia
- Assess gait –
    - Short step
    - Stooped posture
    - Difficult to initiate, stop and turn
    - Less arm swing

Relevant:

- Olfaction
- Minimental status (Lewy body dementia)
- Cerebellar signs (Shy Dragor syndrome)
- Vertical gaze abnormality (Supranuclear palsy)
- Postural drop (ShyDragor syndrome)
- Side effects of dopamine (abnormal movements)

1.10: Scheme for speech examination

Speech examination

Can be?

- **Dysphasia**

  OR
- **Dysarthria**

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For dysphasia examine:

1. Fluency
2. Comprehension
3. Naming
4. Repetition
5. Reading
6. Writing
7. Calculation

Relevant Examination:

- Facial weakness
- Hemiplegia
- Homonymous hemianopia

Followed by examination of:

- Blood pressure
- Pulse
- Carotid and precordial auscultation
- Xanthelasmas
- Bed sores
- Gait

Note: for dysartheria follow cerebellar lesion scheme

1.11: Explanation of hemiplegia/hemiparesis

Middle age man lying on bed with branula on his Right hand. On examination o lower limb, bulk is B/L normal & comparable. There are no spontaneous or inducible fasciculation. There is hypertonia along with grade III hyper reflexia in left lower limb Power is 0/5 in both proximal & distal muscles on left side along with extensor Plante response.

Similarly there is hypertonia & grade III hyperreflexia in left upper limb & powe is 0/5 in both proximal & distal muscles.

There is left sided facial nerve palsy of upper motor neurontype. Rests of th cranial nerves are grossly intact. Cerebellar signs & gait cannot be assessed because o reduced power.

Patient has regular pulse of good volume with no radio-radial delay blood pressure in 160/100mm Hg. There is no carotid bruit or murmur on precordial auscultation. There are no bed sores & lung fields are clear to auscultation. There are no xanthelasmas or corneal arcus.

My impression on these clinical findings is Left sided hemiplegia most probably due to cerebral ischemia at the level of internal capsule due to occlusion of lenticulo-striate arteries.

Viva questions:

What are differential diagnosis?

Differential diagnosis of stroke include:

- Space-occupying lesions
- Cerebral tumour
- Abscess
- Parasitic e.g. neurocystercercosis, echinococcus (hydatid disease)
- Viral encephalitis
- Neuroinfl ammatory
- Demyelinating plaque in MS
- Neurosarcoid
- Behçet's
- Metabolic: hypoglycaemia.
- Migraine: may present with aphasia and hemiparesis.
- Epilepsy: Todd's paresis following a focal seizure.

How to investigate?

Immediate:

- Oxygen saturations, glucose, BP, 12-lead ECG, blood tests (FBC, clotting, ESR, TFTs, LFTs, ANA, blood cultures).
- CT brain (often normal in the acute phase in embolic stroke) –may see thrombus (e.g. in proximal middle cerebral artery) or the hyperdense signal of blood in haemorrhagic stroke.

Urgent:

- CXR (to rule out aspiration).
- Fasting glucose and lipids.

Differential diagnosis include:

- GBS
- CIDP

Viva questions:

How would you investigate?

Blood:

- CBC/ inflammatory markers
- RFTS/LFTS
- Blood glucose and HbA1c
- Vitamin b12/folate
- TFTs
- Protein electrophoresis
- Autoimmune screening

Other:

- Urine for glucose and bence jones proteins
- Nerve conduction studies
- CSF analysis
- Nerve biopsy
- Genetic testing

1.14: Explanation of Parkinson's disease

This patient has mask like expressionless face with infrequent blinking and low volume, monotonous, tremulous speech. There is tabulation of head. When eyes ar gently closed, there are tremors of eyelids (blephroclonus). There is continuous pi rolling tremors of hands with cogwheel rigidity at wrist and lead pipe rigidity at elbow. The rigidity in one limb increased by voluntary movements of the opposite limb. There is bradykinesia with paucity and decrease amplitude of movements. The signs demonstrat asymmetry with the tremors and rigidity more marked on the right side.

Likely diagnosis is Parkinson's syndrome. I would like to check BP with postural drop, and assess cognitive functions and hand writing if not done already.

Viva questions:

How would you investigate?

It's a clinical diagnosis

Brain imaging is usually done to rule out other conditons

How to treat?

Drugs:

- Dopaminiergic drugs
    - Levodopa
    - Dopamine agonist- carbergoline/bromocriptine
    - MAOI-B – selegline
- Anticholinergic – procyclidine/benztropine
- COMTI – tolcapone/entacapone
- Apomorphine – given subcutaneously if can't take oral medications.
- Amantadine

Surgical – deep brain stimulation/thalamotomy/pallidotomy/subthalamotomy

1.15: Explanation of Cerebellar syndrome:

On examination of upper limb there is dysdiadochokinesia, impaired past pointing and intentional tremors that become worse as finger approach target on right side. There is failure of right displaced limb to find its original position with the eye closed. On examination of lower limbs there is impaired heel shin test on the right side along with hypotonia on same side. There is pendular knee jerk. There is Nystegmus with fast component towards right side. There is loss of smooth pursuit. The saccadic eye movements are abnormal with hypermetric saccades. No sign of intra-nuclear ophthalmoplegia. The speech is slurred and has explosive character. The gait is broad based and ataxic with tendency to fall towards right. Romberg test is negative.

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Chapter –

Short Case Examination in Abdominal System

2.1: Scheme for abdominal examination

Command: Examine Abdomen & Do relevant

- Introduction & consent
- Position (Supine, hands by side).
- Proper exposure
- From foot end, take a keen look for 30 seconds from head to toe and try get as much information as possible like; generalized wasting, Bran position, Foley's, Pigmentation, dialysis fistula, abdominal disten surgical scars, stomas, gastrostomy tube, peritoneal dialysis catheter an any additional clues, such as those around the bedside such as walkin adds/special shoes/artificial tears or saliva/glucometer/insul syrings/inhalors etc.
- Now from right side bend till at patient level & observe for any peristals or pulsation.
- Take a look at opposite flank for fullness.
- Ask Patient to cough and check for hernial orifices (Umbilical an inguinal) take consent before checking inguinal.
- If visible prominent veins - check flow.
- Look closely for Striae, (Color, Orientation) any scar mark especially fo paracenteses and Dressing,

Palpation and percussion:

- Ask if any pain in abdomen and tell them if there is any tenderness the should tell you at which point you will stop.
- Keep looking towards patient facial expression during whole of palpation
- Perform superficial palpation to assess any tenderness.
- Bend yourself a bit and keep arm straight at level of your palm duri whole palpation.
- Perform Deep palpation to assess any masses (size, location, consisten mobility, tenderness).

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- Perform visceral palpation.
- For hepatomegaly- Assess the liver by palpation using the edge of the hand from the RIF to the right hypochondrium. Ask the patient to breathe in and out, moving the leading edge of the hand up to feel downward motion of the liver edge during inspiration. Check for span, surface, tenderness, pulsation, consistency, and border.
- Percuss upwards from the RIF in the midclavicular line until you reach liver dullness. Percuss downwards in the right midclavicular line until you reach liver dullness. The upper border of the liver is usually at the level of the 5th intercostal space. Measure the distance between the 2 points of dullness with a tape measure or by the number of finger breadths. The normal liver is usually not larger than 12–15cm in the midclavicular line.
- For splenomegaly- from the RIF to the left hypochondrium. Use the tips of the fingers for palpation, advancing them on inspiration. Check for span, surface, tenderness, pulsation, consistency, and border.
- Percuss for the spleen from the RIF diagonally to the left hypochondrium. In normal individuals the percussion note should remain resonant throughout.
- Bimanual palpation for Kidneys bilaterally: Put the hand in the costophrenic angle posteriorly and palpate downwards with the hand on the anterior abdominal wall. A ballotable kidney will usually indicate renal enlargement.
- Bladder palpation for enlarged/obstructed bladder. Don't forget to percuss for dullness in this area as well.
- Palpation of Para aortic Lymph nodes
- Inguinal Lymph nodes (after taking consent).

Percussion:

- Check for Shifting Dullness and Fluid Thrill.

Note:

- **Confirm presence of liver & Spleen with percussion too.**
- **Percuss upper border of liver & measure span, even if you are not able to palpate lower border.**
- **If you are unable to palpate spleen, then you can check it again with palpation while turning patient towards you during testing for Shifting Dullness (keeps you from turning patient again and again)**

Auscultation:

- Listen for bowel sounds, best heard in RIF.
- Hepatic & splenic bruit if they are enlarged.
- Renal bruit ~ 4cm diagonally above the umbilicus.
- Cover abdomen and proceed to relevant examination.

Relevant examination:

- Hands and nails
    - Check for Flapping Tremors on outstretched hands.
    - Check clubbing by coming to level of hand with help of card.
    - Look for nail changes such as leuconychia, half and half n (proximal half of the nails white, the rest darker with a sh demarcation between then), Mees' lines (transverse white li across the fingernail without pitting) and Beau's lines (transve pitted lines across the fingernails) can all indicate renal disease
    - Look for pallor & Jaundice.
    - Palmer crease pigmentation(addisons) /vitiligo
    - Blood sugar check prick mark
    - Palmar erythema
    - Dupuytren contracture (alcoholic cirrhosis)
- Arms
    - Feel pulse and ask for BP
    - Check if any Medical alert bracelt
    - Bruises, petechias at forearm
    - Tattoo marks or IV marks (drug users)
    - An arteriovenous fistula may be present in patients haemodialysis.
    - Excoriation marks (cholestatic/obstructive jaundice).

Face:

- Eyes –Gently pull down the eyelid and look for anaemia with the pati looking up, then for jaundice with the patient looking down. It is suffici to pull down only one eyelid!
- Parotid enlargement (alcoholic cirrhosis)

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- Lips and mouth— oral ulcers/hyperpigmentations (addisons)/ telangiectasia/macroglossia (acromegaly or amyloid) /candidiasis/ hairyleukoplakia (HIV)/poor denitition/gum hypertrophy (AML or cyclosporine)
- Neck and back:
  - Spider Navi (On upper chest too)
  - Juglur vein- Hepato-jugular reflux (right heart failure)
  - Kyphosis/scoliosis
  - Postrolateral renal scar for nephrectomy or adrenelectomy scars
  - Sacral edema
  - Cervical lymphnodes if dectected then axillary lympnodes
  - Acanthosis nigricans
- Gynecomastia
- Pedal edema bilaterally (Check against bone & while looking at patient face) and note if it is unilateral (venous or lymphatic obstruction) or bilateral (heart failure, renal failure, hypoalbuminaemic conditions, nephrotic syndrome) and demarcate the extent of the oedema.
- Say thanks & Cover them up.
- Tell the examiner how you would complete your examination by assessing the hernial orifices (if not done), the external genitalia, performing a rectal examination and looking at the observations chart. I would also like to see urine dipstick for glycosuria and looking at the fundi if the patient has features of end-stage renal disease (ESRD) or diabetes mellitus.

2.2: Explanation of abdominal case

Case # 1

Middle aged emaciated man lying on bed comfortably with Branula on right forearm. On abdominal examination, abdomen in distended with bilateral Flank Fullness. Umbilicus is central and slit like. There is a dressing on left Flank, with underlying puncture wound, However Dressing is Dry and not soaked with fluid or blood with no signs of infection. There are prominent vein with flow directed away from umbilicus.

There is non-tender hepatomegaly, almost 3 finger breath below rightcostal margin, with total span of 18cm in right mid clavicular line, with smooth surface, rounded border, and firm consistency & non-pulsatile. There is palpable splenomegaly, almost 4cm from left costal margin, moving with Respiration & I was not able to get above the swelling. It is non- Tender, having smooth surface, rounded border, firm consistency. There is no palpable accessory lymphadenopathy.

There is evidence of free fluid in abdomen with shifting dullness and fluid Thrill however no eveidence of raised venous pressure or pedal edema.

Gut Sounds are audible with No hepatic or splenic Bruit.

On extending my examination, patient is pale and mildly jaundiced, with multiple spider navi on trunk, petechias and bruises on both forearm. There is no asterexis or signs of ecephalopathy and No evidence of any Rheumatological Diseases.

My Clinical Diagnosis based on these findings is Decompensated Chronic liver disease with signs of portal hypertension, and most probable cause would be viral hepatitis.

I would like to complete my examination by assessing the hernial orifices (if not done), the external genitalia, performing a rectal examination and looking at the observations chart. I would like to do urine dipstick and fundoscopy.

Other differential:

- ____________________?
- ____________________?
- ____________________?

(Think)

Viva questions

Can you name some complications of CLD?

Complications of CLD include:

- Portal hypertension

- Haemorrhage, usually upper GI
- Ascites
- Spontaneous bacterial peritonitis
- Hepatic encephalopathy
- Hepatorenal syndrome
- Hepatopulmonary syndrome.

How do you grade severity of hepatic encephalopathy?

Hepatic encephalopathy is graded according to the West Haven criteria:

- Grade 0: clinically normal but with small changes in memory or mentation.
- Grade 1: mild confusion, euphoria or depression; short attention span and impaired mental tasks such as addition; disordered sleep patterns.
- Grade 2: drowsiness, lethargy, mild disorientation mainly for time but also sometimes for place; inappropriate behaviour and personality change; impaired mental tasks such as subtraction.
- Grade 3: somnolent but rousable, usually with verbal stimuli sufficing; grossly confused and disorientated; speech may be incomprehensible; amnesia often present.
- Grade 4: comatose; no response to verbal or painful stimuli.

How do you classify causes of jaundice?

Jaundice can be classified as:

- Pre-hepatic– 'due to excessive breakdown of red blood cells e.g. autoimmune haemolytic anaemia, malaria, hereditary haemoglobinopathies (e.g. sickle cell disease).
- Hepatic– due to hepatocyte injury e.g. acute viral hepatitis, paracetamol, alcohol, hypoxic/ischaemic.
- Post-hepatic– due to obstruction to the normal flow of bile e.g. gallstone obstruction, pancreatic head malignancies.

How do you manage ascites in association with CLD?

- Salt restriction (90mmol or 5.2g salt/day).
- Spironolactone from 100–400mg/day, in all patients able to tolerate the drug without excessive hyperkalaemia.
- Loop diuretics should be initiated next, usually furosemide from 40–160mg/day.

- If these measures fail or if the patient has significant symptoms related to th ascites, therapeutic paracentesis should be carried out.

- Radiological procedures such as transjugular intrahepatic portosystemic shu (TIPS) procedure may help to relieve portal hypertension but the risk encephalopathy is increased as nitrogenous waste effectively bypass the liver wi a resultant increased concentration in the systemic circulation.

- Surgical measures (portosystemic and peritoneovenous shunts) are much le frequently used these days but still play a role in recurrent ascites. Liv transplantation is a curative procedure for end-stage liver disease when medic managementfails.

Case # 2

Ascites:

On examination of abdomen, abdomen is distended with bilateral flankfullness, howev venous pressure is not elevated and there is no pedal edema. There is hepatosplenomegaly or lymphadenopathy. There are no stigmata of chronic liver disea with no asterixis or signs of encephalopathy.

I would like to complete my examination by assessing the hernial orifices (if not done the external genitalia, performing a rectal examination and looking at the observatio chart.

Most likely patient is having ascites and most common differentials include:

- Infective – Tuberculosis
- Malignant – Peritoneal carcinomatosis/ Lymphoma
- Vascular – portal vein thrombosis/buddchiari syndrome/CCF
- Metabolic – hypoalbuminemia due to cirrhosis/CRF/malabsorptio myxedema

Ascites with Splenomegaly and Jaundice:

Differential Diagnosis:

- Decompensated Cirrhosis with portal hypertension.
- Acute on Chromic liver Disease.
- Hepatoma.
- Disseminated Tuberculosis
- Metastatic
- Lymphoma with peritoneal metastasis.

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- Systemic lupus Erythromatosis.

Ascites with Splenomegaly:

Differential diagnosis:

- Decompensated chronic liver disease
- Lymphoma
- Systemic lupus Erythromatosis
- Disseminated tuberculosis
- Portal vein thrombosis

Tender hepatomegaly with Ascites:

Differential diagnosis along with relevant you need to look for:

- Hepatoma / metastatic (irregular Surface, any lymph. node)
- Alcoholic cirrhosis. (Parotid enlargement, Dup. contracture)
- Acute on Chronic liver Disease (deep Jaundice)
- CCF, constrictive pericarditis (Auscultate heart, JVP)
- Lymphoma (Lymph Nodes)

Ascites with pedal edema:

Differential diagnosis:

- Decompensated Chronic Liver Disease (Stigmata of CLD)
- Congestive Cardiac Failure with hepatic congestion. (JVP and Precordium)
- Nephrotic Syndrome (Periorbital puffiness).
- Malabsorption (Petechia, bruises, Proximal muscle weakness, anemia)

Viva questions:

How would you investigate?

- CBC with inflammatory markers to exclude infection
- LFTS including albumin
- Coagulation profile
- Urea and electrolytes
- Diagnostic paracentesis
- Urine dipstick for protein losses
- Viral and autoimmune screen

- Abdominal USG

How would you distinguish between transudative and exudative ascites in patien with low serum albumin?

Use the serum: ascites albumin gradient (SAAG). A low gradient (<1.1g/dL indicates loss of protein into the ascites and is a sign of an exudate whereas a hig gradient (>1.1g/dL) indicates a transudate.

Case #3

Hepatomegaly:

On examnation of abdomen there is tender/non tender hepatomegaly 4 finger brea below right costal marging, with smooth surface and no hepatic bruit. There is n associated splenomegaly or lymphadenopathy. There is no clinical eveidence of ascite and venous pressure is not elevated with no pedal edema. There are no stigmata of CL Additional findings may include cachexia, pallor and jaundice.

I would like to do external genitalia examination, digital rectal examination and urin dipstick.

Most likely differential includes:

Infective casuse: viral hepatitis/ malaria/liver abcess /hydatid disease

Vascular: budd chiari/constrictive pericarditis/CCF

Inflammatory: primary blliary cirrhosis

Infiltrative: metastatic/amyloidosis/sacrcoidosis

Metabolic: alcoholic/hemochromatosis/NAFLD

Neoplastic: hepatoma/ metastatic /CML

Other: polycystic liver disease

Note:

- **Hepatomegaly in the absence of stigmata of CLD is unlikely to be due to cirrhosi Consider a malignancy instead.**
- **Chronic myeloid leukaemia may present with jaundice secondary to a autoimmune haemolytic anaemia. The jaundice is pre-hepatic, giving the sclera lemon yellow tinge.**
- **Take care not to confuse the rectus sheath with a liver edge! Avoid this palpating just lateral to the rectus sheath.**

Differential diagnosis of Tender Hepatomegaly:

Young

- Acute viral hepatitis.
- Liver abscess.
- Alcoholic hepatitis.
- CCF/Constrictive pericarditis.
- Budd Chiari Syndrome

Old

- Hepatoma/metastatic.
- And all as in young.

Differential diagnosis of Non Tender Hepatomegaly:

Small Size

- Enteric Fever
- Malaria (Chronic)
- Chronic Myeloid Leukemia.
- Lympho-proliferative Disorder.
- Sarcoidosis.

Huge

- Hepatoma
- Metastatic
- Hydatid Cyst.
- Polycystic Liver.

Non Tender Hepatomegaly with Jaundice:

Differential diagnosis:

- Wilson's Disease
- Primary biliary cirrhosis
- Autoimmune
- Hemochromatosis
- Lymphoma.

Viva questions:

What are the biochemical features of Wilson's disease and what are the limitations of these tests?

- Low serum caeruloplasmin and high urine copper levels are the cardinal features of Wilson's disease.
- Both copper and caeruloplasmin are acute phase reactants and their levels increase in the presence of an acute phase response. Therefore, levels should be interpreted in the context of the patient's overall condition.

What are the most frequent presenting complaints of PBC?

- PBC is asymptomatic in many patients and is diagnosed incidentally e.g. at cholecystectomy.
- In those who present with symptoms, pruritis and fatigue are the commonest presenting features.
- Some patients experience right upper quadrant pain.
- Impaired bile production leads to impaired absorption of fat-soluble vitamins and easy bruising in some subjects.

Can you name some conditions that might mimic PBC?

- The most common differential diagnoses for PBC are autoimmune hepatitis and primary sclerosing cholangitis.
- PBC can occasionally be mistaken for Wilson's disease as Kayser–Fleischer rings may occur as a result of impaired copper excretion.
- Granulomas in the liver may suggest cholestatic sarcoidosis but AMAs are usually absent in this condition.

What treatment options are currently available for PBC?

- General measures include avoidance of alcohol and supplementation of the diet with the fat soluble vitamins (A, D, E, K).
- Ursodeoxycholic acid reduces cholestasis and improves liver function tests. However, it has little effect on symptoms or prognosis.
- Pruritis is generally assumed to be caused by circulating bile acids and may be helped by the anion exchange resin cholestyramine. There is, however, evidence that the itch may have a central component.
- In advanced cases, a liver transplant may be successful. Functional decline, uncontrolled itching, hepatic encephalopathy, bleeding varices and recurrent infections are all indications for transplantation.

How would you treat someone with primary haemochromatosis?

- The mainstay of treatment for patients is to avoid alcohol and iron supplementation in their diet.
- Weekly phlebotomy is still the most reliable way of reducing body iron stores.
- Desferrioxamine, an iron chelator, can be used as injections to reduce iron stores.
- In those with advanced liver cirrhosis or with hepatocellular carcinoma a liver transplant is indicated if the patient is fit enough to undergo the procedure.

Case # 4:

Splenomegaly:

Splenomegaly with anemia –

On abdominal examination this patient is having non tender splenomegaly 5 finger breath below left costal margin, moving with respiaration and I couldn't get above the swelling. There no hepatomegaly or lymphadenopathy. Patient is pale with or without jaundice. There is no clinical evidence of ascites/pedal edema and venous pressure is not elevated. There are no signs of portal hypertension or CLD or rheumatological disease.

I would like to do external genitalia examination, digital rectal examination and urine dipstick.

Considering above mentioned finding most likely differential include:

Hematological: myeloproliferative disorders/hemolytic anemia/ chronic malaria

Inflammatory: Sarcoidosis/SLE/felty syndrome

Infective: tuberculosis/HIV/infectious mononeucleosis

Splenomegaly with lymphadenopathy-

On abdominal examination this patient is having non tender splenomegaly 5 finger breath below left costal margin, moving with respiaration and I couldn't get above the swelling. There is associated non tender hepatomegaly 3 finger breath below right costal margin, with smooth surface and no bruit. There is cervical lymphadenopathy. Patient is pale with or without jaundice. There is no clinical evidence of ascites/pedal edema and venous pressure is not elevated. There are no signs of portal hypertension or CLD or rheumatological disease.

I would like to do external genitalia examination, digital rectal examination and urine dipstick.

Considering above mentioned finding most likely differential include:

Infective: tuberculosis/HIV/infectious mononeucleosis/toxoplasmosis

Hematological: lymphprolifertaive disorders/leukemia (CLL)

Inflammatory: sarcoidosis/SLE

Hepatosplenomegaly with no lymphadenopathy:

Causes include-

- Infective (as above + Kalazar)
- Myeloproliferative and lymphoproliferative diseases
- Cirrhosis with portal hypertension.
- Wilson's disease
- Haemochromatosis
- Glycogen storage disorders

Top Tips

- If other stigmata of chronic liver disease are present, the diagnosis is likely to be cirrhosis with portal hypertension.
- Ascites does not usually occur in myeloproliferative and lymphoproliferative disorders.
- Lymphadenopathy elsewhere makes the diagnosis of lymphoproliferative disorder more likely.
- Glycogen storage diseases are rare causes of hepatosplenomegaly.

Viva questions:

What is the characteristic chromosomal abnormality in CML?

The Philadelphia chromosome is the hallmark of CML, where it is found in > 90 %of cases. Cytogenetically it usually results from a chromosomal translocation: t(9;22)(q34;q11).

Tell me about Felty's syndrome

Felty's syndrome is defined by the presence of 3 conditions: rheumatoid arthritis, splenomegaly and neutropenia. It affects <1 %of patients with RA.

How would you differentiate between a splenic and renal mass?

The 4 characteristics of a splenic mass are as follows:

- Dull to percussion
- Not ballotable
- Palpable splenic notch
- The palpating finger cannot get above a splenic mass

These features are absent with a renal mass.

Case # 5:

Renal abdomen:

1. On examination this patient is having a scar in RIF with smooth, firm and non tender mass below it with dull percussion note over it with no bruit. There is scar mark on upper chest likely due to previous vascular access for hemodialysis. There is non functioning/functioning left brachiocephalic fistula with no recent signs of needling. There is no asterexis or other signs of uremia. Patient is euvolemic as venous pressure is not elevated with no pedal edema. There is gum hypertrophy likely due to immusuppressive therapy. There blood glucose check marks on finger along with lipodystrophy on insulin injection sites.

I would like to request BP, urine distick and to do heart auscultation for uremic bruit.

Most likely diagnosis is patient is having renal replacement therapy in form of functional renal transplant and likely on ciclosporin for immunosuppression. Likely etiology for renal failure would be diabetic nephropathy.

2. On examination patient is middle aged man, looks mildly pale with few bruises. There is functioning brachicephalic fistula on left arm with signs of recent of needling. (Or there is tunneled CV catheter in right subclavian vein with no signs of infections). There is no asterexis or other signs of uremia. Patient is euvolemic as venous pressure is not elevated with no pedal edema.

I would like to request BP, urine distick and to do cardiac auscultation for uremic bruit.

Most likely diagnosis is patient is having renal replacement therapy in form of hemodialysis from functioning fistula. Most likely underlying etiology for renal disease can be glomerulonephritis.

Viva questions:

What are the commonest causes of ESRF?

- Diabetes mellitus
- Chronic glomerulonephritis
- Hypertension
- ADPKD

What are the complications of transplanting a patient with Alport's syndrome?

Patients with Alport's syndrome have a defect in type IV collagen, so a new transplant exposes them to antigens they have not encountered before. The subsequent antibody response against this antigen may result in some patients developing anti-glomerular basement membrane disease (the antibody responsible for Goodpasture's disease).

Can you offer some differential diagnoses for that mass in the right iliac fossa?

Common:

- Caecal carcinoma
- Crohns disease

Uncommon:

- Ovarian tumour (in a female)
- Ileocaecal abscess (e.g. with Yersinia sp.).

Can you name some opportunistic infections associated with renal transplantation?

- Cytomegalovirus
- Pneumocystis jiroveci (carinii)
- Epstein–Barr virus (particularly in patients with post-transplant lymphoproliferative disease)
- BK virus (a common polyomavirus which lies dormant in the renal tract after primary infection and can be reactivated by immunosuppression. Complications of BK virus in transplant patients include ureteric stenosis and interstitial nephritis)

Why are kidney transplants placed in the pelvis?

- Good blood supply.
- There is sufficient space for the kidney.
- Proximity to the bladder for the ureteric anastomosis.
- Easy access to perform renal biopsies or nephrostomies (in the event of a ureteric stricture) without having to puncture the peritoneum or risk abdominal viscerae.

What is the commonest cause of death in a patient with a renal transplant?

The commonest cause of death in all patients with renal disease is accelerated cardiovascular disease (CVD). After CVD, the commonest causes of death are malignancy and infections. Both are related to chronic immunosuppression.

Chapter – 3

Short Case Examination in Precordial System

3.1: Scheme for Examination of precordium

Command: Examine Precordium and do relevant:

- Introduction & Consent.
- Proper Position.
- Exposure (Shirt off ideally) if female patient then ask for full exposure but you can be allowed to proceed with limited exposure.
- Take a look from foot end.
  - Head to Toe (Try not to miss any important finding)

    Start by looking for signs of syndromes with associated cardiovascular disease, for instance; Down's syndrome (low-set ears, epicanthic folds, flat nasal bridge), Ankylosing spondylitis (question-mark posture, restricted neck movements), Marfan's syndrome (tall, arachnodactyly, arm span > height), Turner's syndrome (webbed neck, cubitus valgus, short stature, infertility).

    Inspect the face for a malar flush (mitral stenosis) or a slate-grey rash in photo-exposed areas (adverse effect of amiodarone).

    Look for arterial (Corrigan's sign – AR) or venous pulsations (C–V waves – TR). Sometimes a slow rising pulse can be detected on inspection.

    Look for signs of current or previous central access or scars on the chest from tunnelled lines. These can be a source of endocarditis (particularly of the tricuspid valve) but may also have been used to administer long-term antibiotics giving a clue that the patient may have suffered endocarditis in the past.

  - Inspect the praecordium for a sternotomy scar. If one is present, immediately glance at the legs to see if there are any scars from vein graft harvesting. Ask the patient to lift up his/her arms so that you do not miss a thoracotomy scar near the axilla or any other pertinent scars. For female patients, ask the patient to lift her breast up so that you can look for an inframammary scar (mitral valvotomy).

Look for scars from current or previous devices such as a pacemaker or automatic implantable cardioverter-defibrillator (ICD).

Note any gynaecomastia (e.g. from digoxin or spironolactone use or indeed both).

Look for any obvious apical pulsation to determine the location of the apex beat. Listen for audible clicks from a prosthetic mechanical heart valve.

- ➢ Ask for pain in precordium or Chest?
- ➢ Palpate & Try to localize apex beat with one finger tip, (demonstrating that you have done so for the examiner's benefit!).
    - o Feel its character (Thrusting / heaving/Tapping).
    - o Count inter costal space and whether medial or lateral to mid clavicular line.
    - o If Thrill, check whether systolic or Diastolic while palpating carotid artery.
- ➢ Palpate Tricuspid, Pulmonary, aortic area and epigastrium.
- ➢ Check Left parasternal heave by bending at patient level.

Auscultation

- Put left hand fingers on carotid during whole Auscultation to relate murmur with carotid Pulsation.
- Listen with Diaphragm at apical area, ask patient to hold breath at end of expiration to check if murmur intensity is increasing during expiration.
- **Ask patient to breath normally** (Don't forget about it).
- Turn Patient to left side, localize the apex and auscultate again.
- Listen with bell, hold breath at end of expiration to check if murmur intensity is increasing during expiration.
- Ask patient to breath normally.
- Check axillary radiation if Systolic murmur is heard at apical area.

Note:
When using the bell, remember to hold it off the chest slightly rather than pressing it o to the chest. Pressing too hard will turn it into a diaphragm, filtering out the very sound you are trying to hear. If you are pressing too hard, you will leave a rim on the patient skin and your fingernails will go pale making it obvious to any attentive examiner.

- Listen with the bell at the left sternal edge for 3rd and 4th heart sounds or pericardial knocks
- Auscultate at Tricuspid area while asking patient to hold breath at end of Inspiration to check if murmur intensity is increasing during inspiration.
- Listen on pulmonary area with diaphragm
  - For 2nd heart sound. Is it normal, loud (suggestive of pulmonary hypertension) or soft?
  - Listen for splitting of the 2nd heart sound if you are familiar with this; if not do not worry, it is not particularly important in the exam
  - If you hear a pulmonary ejection murmur, check whether it radiates to the back and whether it gets louder with inspiration to ensure you are not mistaking AS for pulmonary stenosis (PS).
- Ask patient to lean forward.
- Auscultate A_1 & A_2 with breath held at end of expiration to check if murmur intensity is increasing during expiration.
- Check carotid radiation in case of Systolic murmur at A1 area (Aortic stenosis).

Note: You should Skip aortic auscultation in lying position to save Time.

- Auscultate lung basis from back while patient is sitting for pulmonary oedema.
- Check for sacral edema while patient is sitting.
- Now position patient at 45°.
- Check Temperature by putting Thermometer in axilla
- With both arms straight and stretched out, feel both radial pulses at the same time to check volume deficit between the 2 sides.
- Assess the heart rate and rhythm at the radial pulse, counting to at least 10 to ensure you are not missing atrial fibrillation.
- Assess the character of the pulse at the brachial artery, determining whether it is normal, slow rising or collapsing. Holding the forearm by the wrist with one hand and supporting the elbow with the other, lift up the arm and forearm so that they are aligned, and straight above heart height.
- Tell examiner you would like to check blood pressure and Radio-radial / radio-femoral delay.
- **Don't Forget To Remove Thermometer At This Stage.**
- Now ask patient to stretch hands in front of you.

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And in hands look for:

- Clubbing by bending at patients hands level and confirm with card (Infectiv endocarditis)
- Splinter hemorrhages (Infective endocarditis)
- Osler nodes (Infective endocarditis)
- Jane way lesions (Infective endocarditis)
- Anemia (Infective endocarditis)

In Forearm look for:

- Petechia
- IV abuse mark

Face – Jaundice, anemia, conjuctival hemorrhage, xanthelasmas, corneal arcus,

Neck – Jugular venous pressure and Hepato-juglur reflux. Turn the head slightly to th left and use a spot light to visualize the JVP. Measure its vertical height above the stern angle. If the patient is in sinus rhythm, there should be 2 pulsations visible: a v-wav with or after the pulse and an a-wave preceding the pulse. Assess their prominence.

Oral cavity – poor dentition, central cyanosis, muller sign

Other –

- Pedal edema
- Planters (To rule out cerebrovascular event due to thrombo-embolism)
- Abdomen- palpate liver and spleen
- Offer funduscopic eye examination for roth spot (Infective endo carditis)

Complete your examination by asking to dip-stick the urine for haematuria an proteinuria (endocarditis) and glycosuria (cardiac risk factor) and checking th temperature chart.

Note: Look for signs of Rheumatological Disorders in case of aortic regurgitation an mitral regurgitation.

Note: In atrial fibrillation, you should count pulse rate for whole one minute whil checking for pulses deficit with examiner's help at same Time. In exam you can't sp one minute on rate count. So you can explain examiner at end if he ask.

Presentation

As a minimum, without having to be asked, you must be able to comment on at least things:

- Which valve(s) and what lesion(s)?
- How bad is it? Or, for a prosthetic valve, is it functioning well?
- Is there any heart failure?
- Is there any evidence of endocarditis (assumed to be present until proven otherwise)?
- What caused it? If there is no obvious cause, speculate from your knowledge of general medicine.

3.2: Explanation of Mitral regurgitation

Young ill looking boy lying on bed in respiratory distress with branula on Right hand. There is no central or peripheral cyanosis. On examination of precordium. There are visible pulsation in left infra-mammary region. However there is no scar mark. Apex beat in palpable in left 6th intercostal space lateral to mid clavicular line, thrusting in character. Pulmonary component of 2nd heart sound is palpable. There is no left parasternal heave or Thrill. On Auscultation1st heart sound is soft, pulmonary component of Second heart sound is loud. There is high-pitched, blowing pan-systolic murmur of grade 3, at apical area, with axillary radiation. Best heard at end of expiration in left lateral position.

On extending my examination pulse in 100 beats/min, regular, good volume & normal Character. Blood pressure is 100/60 mm Hg with 40 pulse pressure. Lung fields are clear to auscultation. Venous pressure is not elevated and there is no pitting ankle or sacral edema.Patient is pale however there are no peripheral stigmata's of infective endocarditis or any evidence of Rheumatological Diseases. Planter response is Downward Bilaterally.

My clinical impression is mitral regurgitation with pulmonary Hypertension. Patient is in sinus rhythm with no evidence of heart failure.

Viva questions:

What are differential diagnosis?

- Mitral regurgitation
- Tricuspid regurgitation
- VSD

What are common causes?

- Degenerative
- Functional (secondary to LV dilatation and causes thereof)
- Ischaemic

- MV prolapse
- Idiopathic
- Marfan's syndrome
- Connective tissue disorder
- Infective endocarditis

How would you investigate?

- 12-lead ECG (may reveal P-mitrale or AF).
- CXR to identify pulmonary congestion and assess cardiac size.
- TTE to assess severity, LV function and dimensions, pulmonary pressures an RV function.
- TOE (to definitively assess severity, define mechanism of MR, feasibility c repair and exclude/diagnose endocarditis) and where available, 3D-TOE.
- Right and left heart catheterization (to assess pulmonary pressures and corona anatomy. If bypass grafts are needed these can be carried out at the same time a valvular surgery)
- Carotid Duplex (to determine whether any carotid disease requires treatment pri to cardiothoracic surgery).
- Orthopentomogram (dental radiograph taken prior to a dental check to ensure an dental extractions required are undertaken before surgery to minimize future ris of prosthetic valve endocarditis).

How would you manage?

Asymtopmatic- regular follow up

Symptomatic – medical management include rate or rhythm control and anticoagula if needed in case of atrial fibrillation. Management of heart failure wi diuretic/ACEI/ARB/Digoxin/B blockers.

What are the indications for surgery?

- Symptomatic NYHA class III/IV despite medical management.
- Asymptomatic with EF< 60% /LVESD > 45mm.

3.3: Explanation of Mitral stenosis:

Young man lying on bed with no respiratory distress with branula on Right han There is no central or peripheral cyanosis. On examination of precordium. There are n visible pulsation or scar marks. Apex beat in palpable in left 5th intercostal space slight lateral to mid clavicular line, tapping in character. Pulmonary component of 2nd heart sound is palpable. There is left parasternal heave, however no Thrill. On Auscultation 1st heart sound is loud, pulmonary component of Second heart sound is also loud.There is low-pitched, rumbling mid-diastolic murmur of grade 3, at apical area, with no radiation. Best heard at end of expiration in left lateral position with bell of stethoscope. There is also a high-pitched, blowing pan-systolic murmur of grade 3, at tricuspid area. Best heard at end of inspiration.

On extending my examination pulse in 100 beats/min, regular, low volume & normal Character. Blood pressure is 100/80 mm Hg with 20 pulse pressure. Lung fields are clear to auscultation. Venous pressure is not elevated and there is no pitting ankle or sacral edema. Patient is pale however there are no peripheral stigmata's of infective endocarditis Planter response is Downward Bilaterally.

My clinical impression is mitral stenosis with pulmonary Hypertension and tricuspid regurgitation. Patient is in sinus rhythm with no evidence of heart failure.

Viva questions:

What are differential diagnosis?

Left Atrial myxoma

Left atrial thrombus

Severe mitral regurgitation (increase forward flow across MV)

How would you investigate?

- 12-lead ECG– may reveal P-mitrale or AF.
- CXR to identify pulmonary congestion and haemosiderosis, and to assess LA size
- Transthoracic echocardiogram (TTE) to assess severity – mean gradient and mitral valve area (MVA); suitability for percutaneous balloon mitral valvuloplasty (PBMV); pulmonary pressures and RV function.
- Transoesophageal echocardiogram (TOE) for detailed assessment of valvular and subvalvular anatomy and to assess LA appendage for thrombus prior to PBMV.
- Right and left heart catheterization to assess coronary anatomy as part of work up for surgery, assess MVA and to assess pulmonary artery systolic (PASP) and wedge pressures (PAWP).

How would you manage?

Asymptomatic – regular follow up with echocardiography

Atrial fibrillation- either rate or rhythm control strategy and anticoagulation if needed.

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Symptomatic- diuretic for symptoms relieve and surgery would be indicated.

What are indication of surgery?

Pulmonary HTN

Pulmonary congestion

Heamoptysis

Recurrent thromboembolism

What are the indications for anticoagulation?

Those in AF (paroxysmal or sustained) should be on warfarin as the risk of thromboembolism is high and > 3-fold higher than in non-rheumatic AF.

Warfarin should be considered in those with LA > 55mm even if in sinus rhythm.

How often should patients with MS be followed up?

Patients who are asymptomatic and have mild MS (MVA > 1.5cm 2 or mean gradien <5mmHg) should be subject to annual follow-up.

What is etiology?

- Rheumatic fever (>90 %of cases).
- Degenerative: severe mitral annular calcification.
- Non-valvular: LA myxoma, ball-valve LA thrombus, large vegetations in infectiv endocarditis.
- Congenital:
- Isolated
- Cor triatriatum (division of one of the atria into 2 by a membrane)
- Shone's syndrome (supravalvular mitral membrane, parachute MV, subaortic stenosis and aortic coarctation)
- Mucopolysaccharidosis
- Left-sided carcinoid syndrome (metastatic or bronchial carcinoid).
- Methysergide or ergot alkaloid valvulopathy (e.g. cabergoline).

How do you grade severity?

Mild: >1.5 cm2

Moderate: 1-1.5 cm2

Severe: <1 cm2

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3.4: Explanation of Aortic stenosis:

Old man laying on bed with no respiratory distress with branula on right hand. There is no evidence of central or peripheral cyanosis. On examination of precordium. There are visible pulsations in left infra-mammary region, however there is no scar mark. Apex beat in palpable in left 5th intercostal space slightly lateral to mid clavicular line, normal in character. There is palpable systolic thrill at aortic area. Pulmonary component of 2nd heart sound is also palpable along with left parasternal heave. On Auscultation 1st heart sound is normal, pulmonary component of Second heart sound is loud. There is harsh ejection systolic murmur of grade IV at aortic area, with radiation to carotid, best beard at end of expiration in leaning forwards position. There is also a high-pitched, blowing pan-systolic murmur of grade 3, at tricuspid area. Best heard at end of inspiration.

On extending my examination pulse in 100 beats/min, regular, low volume & normal Character. Blood pressure is 100/80 mm Hg with 20 pulse pressure. Lung fields are clear to auscultation. Venous pressure is not elevated and there is no pitting ankle or sacral edema. Patient is pale however there are no peripheral stigmata's of infective endocarditis. Planter response is Downward Bilaterally.

My clinical impression is aortic stenosis with pulmonary Hypertension and tricuspid regurgitation. Patient is in sinus rhythm with no evidence of heart failure.

Viva questions:

How would you investigate?

- ECG- may show LVH, left atrial enlargement
- CXR-
- Echocardiography to confirm the diagnosis and grade the disease in terms of severity, with severe AS being <0.6cm 2/m 2
- Stress echocardiography - can be very useful in the presence of poor LV function to decide whether the impaired LV is due to severe AS and therefore may benefit from surgery, or if the main problem is due to intrinsic myocardial disease with incidental and non-contributory AS, which does not improve with aortic valve replacement.
- Cardiac catheterization- coronary angiography will be necessary prior to surgery to see associated CAD

How would you manage?

If asymptomatic, it is generally recommended that 6–12-monthly followup is sufficient.

Some would recommend surgery if the stenosis is severe, even in the absence of symptoms. The following criteria are typical:

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- Consider operating if
  - Mean gradient > 40mmHg and any of:
  - LV systolic dysfunction i.e. ejection fraction (EF)<45 %
  - Abnormal response to exercise (dBP)
  - Ventricular tachycardia
  - LVH > 15mm
  - Valve area <0.6cm 2

How do you grade severity?

- Mild - Valve area > 1.5cm 2
- Moderate - 1–1.5cm 2
- Severen - <1cm 2

What are the common complications of AS?

- Endocarditis
- Heart failure
- AV block due to invasion of calcium from the valve ring into the His-Purkinje system
- Embolic events.

How would you differentiate sclerosis from stenosis?

Normal pulse character, normal 2nd heart sound, frequently in an elderly person. Not this overlaps with mild AS.

What are the associated conditions?

- Coarctation of the aorta (remember to check for radio-femoral delay).
- Other valvular disease, particularly mitral in rheumatic valvular disease.
- Angiodysplasia of the colon and anaemia.

3.5: Explanation of Aortic regurgitation:

Middle aged ill looking man lying on bed in respiratory distress with branula on Rig hand. There is no evidence of central or peripheral cyanosis.On examination (precordium. There are visible pulsation in left infra-mammary region. However there no scar mark.Apex beat in palpable in left 5th intercostal space lateral to mid clavicul line, heaving in character. No other sounds are palpable and there is no left parastern heave or Thrill. On auscultation 1st and second heart sounds are normal.There is hig pitched early diastolic murmur of grade III at left parasternal area, best heard at end of expiration in learning forward position.

On extending my examination pulse in 100beats/min, regular, good volume & collapsing in Character. Blood pressure is 100/40 mm Hg with 60 pulse pressure. There are prominent pulsation in neck, nail bed capillaries & in uvula and pistol shot femoral. Lung fields are clear to auscultation. Venous pressure is not elevated and there is no pitting ankle or sacral edema.Patient is pale however there are no peripheral stigmata's of infective endocarditis or any evidence of Rheumatological Diseases.Planter response is Downward Bilaterally.

My clinical impression is aortic regurgitation. Patient is in sinus rhythm with no evidenc of heart failure or pulmonary hypertension.

Viva questions:

How would you investigate?

- ECG
- CXR
- Echocardiography
- Cardiac catherization

How would you manage?

- If AR is acute, then if the severity is anything more than mild, surgery is the treatment of choice.
- In chronic AR, if there are symptoms, i.e. anything worse than class I breathlessness or anginal chest pain, then again valve replacement should be offered.
- Generally in those with asymptomatic AR conservative medical management with ACE inhibitors and diuretics is indicated.

Surgery is indicated even if asymptomatic with any of the following:

- LV dilatation LVEDD >70, LVESD> 50.
- EF <50 %
- Significant aortic root dilatation (maximal root diameter > 45mm in Marfan's. > 50mm with bicuspid valve or > 55mm otherwise)
- Those undergoing any other cardiac surgery

What are complications of AR?

- Endocarditis
- Heart failure

What are the possible aetiologies of AR?

Acute AR

- Infective endocarditis
- Aortic dissection
- Prosthetic valve failure
- Ruptured sinus of Valsalva (rare)
- Acute rheumatic fever (rare in the West, but not elsewhere)

Chronic AR

- Bicuspid aortic valve
- Marfan's syndrome
- Aorto-annular ectasia
- Rheumatic heart disease
- Endocarditis
- Seronegative arthritides
- Syphilis (now rare)
- Osteogenesis imperfecta (very rare)

What clinical findings would suggest severe AR?

- 3rd heart sound
- Signs of left-sided heart failure
- Very wide pulse pressure
- Short murmur

3.6: Explanation of Mitral regurgitation with infective endocarditis:

Young ill looking girl lying on bed in respiratory distress with branula on Right hand There is no central or peripheral cyanosis.On examination of precordium due to limited exposure I cannot comment on visible precordial pulsation or scar mark. However, apex beat in palpable in left 6th intercostal space lateral to mid clavicular line, thrusting in character. Pulmonary component of 2nd heart sound is palpable. There is left parasternal heave and systolic thrill in tricuspid area. On Auscultation 1st heart sound is soft pulmonary component of Second heart sound is loud.There is high-pitched, blowing pan-systolic murmur of grade 3, at apical area, with axillary radiation. Best heard at end of expiration in left lateral position. There is also a high-pitched, blowing pan-systolic murmur of grade 3, at tricuspid area. Best heard at end of inspiration.

On extending my examination pulse was irregular, good volume & normal Character. Blood pressure is 100/50 mm Hg with 50 pulse pressure, temperature was 101 F.There are fine crepitation at lung basis bilaterally. Venous pressure is elevated 4 cm above the right sternal angle. And there is pitting ankle edema with no sacral edema. Patient is pale and clubbed. Splinter hemorrhages seen on nails. Splenic tip was palpable. However there is no clinical evidence of Rheumatological Diseases.Planter response is Downward Bilaterally.

My clinical impression is patient is suffering from infective endocarditis with mitral and tricuspid regurgitation with signs of pulmonary Hypertension, heart failure and probably atrial fibrillation.

3.7: Explanation of Ventricular septal defect:

Young boy lying on bed in respiratory distress with branula on Right hand. There is no central or peripheral cyanosis.On examination of precordium. There are visible pulsation in left infra-mammary region. However there is no scar mark. Apex beat in palpable in left 5th intercostal space in the mid clavicular line, normal in character. Pulmonary component of 2nd heart sound is palpable. There is left parasternal heave and systolic Thrill.On Auscultation 1st heart sound is normal, pulmonary component of Second heart sound is loud. There is high-pitched, blowing pan-systolic murmur of grade 4, at left parasternal area, with no radiation and no effect of respiration.

On extending my examination pulse in 100beats/min, regular, good volume & normal Character. Blood pressure is 100/60 mm Hg with 40 pulse pressure.Lung fields are clear to auscultation. Venous pressure is not elevated and there is no pitting ankle or sacral edema.Patient is pale however there are no peripheral stigmata's of infective endocarditis.Planter response is Downward Bilaterally.

My clinical impression is ventricular septal defect with left to right shunt with pulmonary Hypertension. Patient is in sinus rhythm with no evidence of heart failure.

Chapter –

Short Case Examination in Respiratory System

4.1: Scheme for Examination of back of the chest and do relevant

Command: Examine back of the chest & Do Relevant

- Introduction and Consent
- Bed side clues – oxygen/inhalors/nebulizors/NIV device/sputum pots/walki aids
- → Position (Hands crossed on opposite shoulder)
- → Exposure (Ask for proper exposure even in female patient)
- Consider patient age, body habitus (short stature; cystic fibrosis) and BMI.
- Is patient short of breath? Observe chest shape & movement simulatneusly. Lo at both supraclavicular regions (lung apices) and axillary areas. (If Any Sc bulge, depression, barrel shape, pectus excavatum/carinatum, kyphoscolio radiotherapy tattoos?)
- Use of accessory muscle, pursed lips, Intercostal indrawing.
- Any audible voices (wheeze/stridor/ hoarse voice)
- Ask for cough (note if dry or productive)
- Ask for tenderness & palpate Chest, while looking at patient Facial expression f tenderness.
- Check Chest expansion at apical area & at back of the Chest and compare if sam bilaterally. Place the palm of your hands with fingers in the intercostal spaces o either side of the patient's chest and pull in the subcutaneous tissues slightly. Li your thumbs off the chest wall so that they meet opposite each other in the midd of the chest. Ask the patient to take a deep breath in and out of their mouth a observe for any asymmetry or reduction in expansion of the chest wall moveme
- Check for Tactile Fremitus and compare bilaterally.
- Start Percussing apical area & move down the back of Chest performi Percussion in intercostal spaces & comparing bilaterally, not forgetting axilla. the percussion note resonant, dull or stony dull? Absence of hepatic and cardi dullness may signify hyperexpansion.

Note: (Inform patient I will hit my finger with my other finger, it wouldn't cause you any pain).

- Similarly Start auscultation from apical area, and move down on back & axillary areas, comparing bilaterally. Consider the following in each area of the chest:
    - Is the air entry normal or reduced?
    - Are the breath sounds vesicular or is there bronchial breathing?
    - Is the inspiratory: expiratory ratio of respiration normal (usually 1:2) or is there a prolonged expiratory phase (airflow obstruction)?
    - Are there any added sounds such as wheeze, crepitations or a pleural rub?
    - Ensure you describe these sounds detailing where they occur in the respiratory cycle as well as their quality, for example, are they fine late inspiratory crepitations or early and course?
- Check vocal Resonance & compare It Bilaterally.

Note:

- (Listen for whole Inspiration & expiration).
- (If crepitation found, ask Patient to cough & Check for change in Character).

Relevant Examination:

- Ask the patient to Lie Down.
- Trachea: Warn the patient that you are going to feel their windpipe and that this may feel slightly uncomfortable. Place your middle finger in the centre of the trachea with your index and ring finger on either side. Look for evidence of deviation or tracheal tug. Check the crico-sternal distance (the distance between the suprasternal notch to the cricoid cartilage). This is normally 3 finger breaths and is reduced in hyperinflation.
- Apex beat: Feel for the patient's apex beat which may be displaced due to mediastinal shift (collapse/massive effusion) or poorly palpable due to hyperexpansion of the chest.
- Pulmonary hypertension: Palpate for a right ventricular heave by placing the palm of your hand over the left sternal border and feel for a palpable pulmonary 2nd heart sound.
- Hands for
    - Palmar erythema (CO2 retention).
    - Wasting of the small muscles of the hand (Pancoast tumour).

  - changes of rheumatological conditions associated with respiratory disea e.g. rheumatoid arthritis, scleroderma

- Nails for
  - Clubbing
  - Peripheral cyanosis
  - Tar staining and yellow nails (yellow nail syndrome).
- Ask the patient to stretch hands for
  - Fine tremor associated with beta 2 agonist use.
  - Course flapping tremor of CO 2 retention
- Check Tenderness at wrist (hypertrophic osteoarthropathy) & sensory loss at T_1 (brachial plexus involvement) if you found clubbing & suspecting apical lu tumor.
- Check for bounding pulses (sign of c02 retention).
- Look at face:
  - A cushingoid appearance suggesting steroid therapy.
  - Stigmata of underlying pathology e.g.: connective tissue disease e. telangiectasia and microstomia in scleroderma patients; butterfly rash i patients with systemic lupus erythematosus (SLE); heliotrope rash dermatomyositis.
  - Facial plethora consistent with secondary polycythaemia – evidence SVC obstruction (facial and upper body swelling)
- Examine eyes for
  - Suffused conjunctivae present in secondary polycythaemia.
  - Horner's syndrome (meiosis, partial ptosis, enophthalmous, anhydrosis).
  - Ask the patient to look up and warn them that you are going to pull dow gently on their lower eyelid to look for anaemia (present in chronic diseas e.g. cystic fibrosis.) and jaundice (which may indicate malignancy).
- Examine mouth for:
  - Evidence of central cyanosis in the lips and the undersurface of the tongue
  - Evidence of oral candidiasis – consistent with steroid use
  - Examine neck for
  - Patient's neck size (larger collar size increases risk of obstructive slee apnoea).

  - Assess the patient's jugular venous pressure (JVP) at 45 °, a raised JVP may suggest cor pulmonale, would be fixed in superior vena cava obstruction (SVCO). "a" wave prominent (right atrial hypertrophy) or large v waves (tricuspid regurgitation)?

- Pedal edema.
- Lymphadenopathy: Ask the patient to sit forward and cross their arms over their chest. Palpate for cervical and supraclavicular lymphadenopathy– ensure you feel all areas (submandibular, pre- and post-auricular, anterior and posterior cervical triangle, occipital and supraclavicular regions). If you find lymphadenopathy offer to examine the axillae.
- Check forced expiratory Time if Suspecting COPD (Keep it to end).
- Check Bony Tenderness at sternum or vertebral bodies where you want for bony metastasis.
- Look for Rheumataogical signs in case of plural effusion.
- Signs of steroid use (rounded face, central obesity, intrascapular fat pad, thin skin, bruising, hirsuitism, proximal muscle weakness)

Ensure your patient is covered up, and thanks the. To complete the examination, offer to look at the patient's observation chart and sputum pot (if one is available), to perform a peak flow/bedside spirometry and to look at the patient's chest radiograph (CXR).

Note: Examination of Respiratory system is lengthy, don't worry if you skip steps in relevant system as far as you have made correct interpretation of your findings with Clinical evidence.

Note: It's not necessary to examine front of the Chest too if you are given command for back of chest. Findings must be on back. Save your Time.

4.2 Explanations of Pleural effusion

Middle age lady of normal built, having branula on right hand, sitting on bed with respiratory distress having Respiratory rate of 24 breaths / min. However there is no evidence of central or peripheral cyanosis. On examination of the back of Chest there is a prominent bulge on the Right infra scapular region. However there is no scar mark. Chest movements are reduced in the Right infra scapular region & chest expansion is less than 3cm. Percussion note is stony dull in the Right infra scapular region. Breath sounds are absent in the same area along with a bronchial breathing just above the area. Tactile fremitus & vocal resonance are reduced in the same area.

On extending my examination trachea is central in position with no tracheal tug & normal crico-sternal distance. Apex beat is not displaced. Finger are not clubbed, patient is not pale neither jaundiced. Venous pressure is not elevated. There is no pitting ankle edem There are no palpable accessory lymph nodes & there are no stigmata of Rheumatologic Diseases.

My Diagnosis based on these clinical findings is Right sided pleural effusion.

Viva questions:

- **What are differenctial diagnosis?**
    - Pleural thickening
    - Consolidation
    - Collapse
    - Raised hemidiaphragm
- **What investigations would you like to do?**
    - Pleural fluid analysis (diagnostic thoracocentesis) Serum levels (album LDH) must be sent simultaneously to apply Light's criteria
    - Serum inflammatory markers (CRP/ESR).
    - Blood tests (FBC, U&E, LFT, TFT, coagulation).
    - Blood cultures (if empyema suspected).
    - Echocardiography (ventricular function, valvular lesions).
    - Radiological
    - CXR
    - Pleural ultrasonography
    - CT thorax
    - Sputum culture (including AFB stain).
    - Mantoux/interferon gamma releasing assay test (TB).
    - Bronchoscopy
    - CT-guided biopsy (when tissue diagnosis required in suspected malignancy
    - Pleural biopsy (useful in suspected malignancy/TB).
- **Outline the management of a pleural effusion and what additi management is required in cases of recurrent effusions?**
    - Supportive measures:
        - Oxygen.
        - IV hydration.

        - Chest physiotherapy (where applicable).
    - Diagnostic ±therapeutic thoracocentesis or chest tube insertion
    - Treatment of underlying cause: Antibiotics (empyema)/Diuretics (CCF).
    - If effusions are recurrent, additional options for therapy do exist:
        - Continuous tube thoracostomy.
        - Pleurodesis (most commonly done in recurrent malignant effusions; can be performed medically with talc or surgically via video assisted thoracoscopic surgery [VATS]).
        - Placement of pleuro-peritoneal shunt (rarely used).

- **What is 'yellow nail syndrome'?**

  It is a rare cause of exudative pleural effusions and is a condition of yellow curved nails associated with lymphoedema, bronchitis, bronchiectasis, sinusitis, nephrotic syndrome and hypothyroidism.

4.3: Explanations of Bronchiectasis

Middle age lady with normal built sitting on bed with Respiratory distress having respiratory rate of 24 breaths/min. On Examination of back of chest is normal in shape with no scar mark. Chest movements are reduced bilaterally & equally. Chest expansion is less than 3cm. Percussion note is resonant throughout the both lung fields. Breath sounds are vesicular with prolonged expiration & there is expiratory wheeze throughout the Chest. There are coarse inspiratory crepitation at both lung basis which are changing character with cough. Tactile fremitus & vocal resonance are comparable bilaterally.

On extending my examination trachea is central in position with no tracheal tug & normal crico-sternal distance. Apex beat in not displaced (may be on Right side if Dextrocardia). Fingers are clubbed, with no evidence of central or peripheral cyanosis. Pulmonary component of second heart sound is loud, however venous pressure is not elevated and there is no pitting ankle edema. There is no palpable accessory lymphadenopathy.

My clinical Diagnosis based on these findings is Bronchiectasis with pulmonary hypertension.

Viva questions

What investigations would you like to offer?

- Laboratory
    - Sputum CS, AFB and cytology.
    - FBC: anaemia of chronic disease, WCC with neutrophilia, polycythaemia secondary to hypoxia.

  - Serum immunoglobulins: IgG subclasses, IgM and IgA (hypogammaglobulinaemia).
  - Alpha-1 antitrypsin levels (alpha-1 antitrypsin deficiency).
  - Aspergillus precipitins and serum IgE (ABPA).
  - Autoantibodies (if connective tissue disease suspected).
- Radiology:
  - CXR: Dilated and thickened airways (tramlines and ring shadows) And Hyperinflation.
  - HRCT– Bronchial thickening and dilatation seen as 'tram tracking' in parallel or ring shadows end on/Signet ring sign/Mucus pluggin (bronchoceles).
- Lung function testing: commonly obstructive impairment
- Others
  - Flexible bronchoscopy
  - Sweat test (chloride > 60mmol/L) and Genotyping for cysti fibrosis (CF)
  - Nasal brushings and electron microscopy for Kartagener' syndrome
  - Tuberculin skin test, interferon gamma release assay fo tuberculosis

How would you manage the case?

- General measures
  - Education of patient and family
  - Optimize nutrition
  - CF patients may need pancreatic enzyme replacement and fat-solub vitamin supplementation.
  - Smoking cessation.
  - Vaccinations e.g. Pneumovax, Hib.
  - Sputum clearance/physiotherapy: Involves postural drainage, che percussion, controlled breathing exercises, and forced expiration.
- Medical treatment
  - Antibiotics to treat infective exacerbations with pseudomonal coverage

- Prophylactic antibiotics e.g. azithromycin orally and/or tobramycin nebulized to cover Pseudomonas species.
- Bronchodilators (if any component of reversibility).
- Steroids– regular inhaled to decrease chronic inflammation and oral in infective exacerbations.
- Mucolytics e.g. carbocysteine, RhDNAase in CF patients.

- Surgical treatment: Lobectomy/bullectomy (note very specific indications– localized disease with good lung function).
- Transplantation in CF.
- Treatment of underlying cause (e.g. immunoglobulins for hypogammaglobulinaemia

What other systems are affected in CF patients?

- Gastrointestinal:
    - Pancreatic enzyme insufficiency with malabsorption of fat and protein.
    - Diabetes (cystic fibrosis-related diabetes– CFRD).
    - Meconium ileus (in newborns) and distal intestinal obstruction syndrome (DIOS).
    - Focal biliary cirrhosis leading to or with portal hypertension.
    - Cholelithiasis.
- Reproductive: male subfertility (> 95 %) due to defective sperm transport. Spermatogenesis is normal.
- Musculoskeletal: osteoporosis.
- ENT: sinus disease and nasal polyposis.

Give the main complications of bronchiectasis?

Pulmonary complications

- Recurrent infection
- Haemoptysis (can be massive, i.e. > 400mL)
- Empyema/abscess
- Cor pulmonale (raised JVP, right ventricular heave, peripheral oedema).

Extrapulmonary complications

- Anaemia
- Metastatic infection e.g. cerebral abscess

- Secondary amyloidosis.

What are the major pathogens associated with bronchiectasis?

- Pseudomonas aeruginosa
- Haemophilus influenza
- Streptococcus (less commonly).

4.4: Explanations of chronic obstructive airway disease

Old age man of normal built, sitting on bed in Respiratory distress wi respiratory rate of 24 breaths/min. Patient is having pursed lips and using accesso muscles of Respiration with obvious Supra clavicular and intercostals recessions.

On examination of back of chest, chest is barrel in shaped, however there is scar mark Chest movements are bilaterally reduced but equal. Chest expansion is le than 3cm.Percussion note is Resonant throughout the both lung fields. Breath sounds a vesicular but of diminished intensity, with prolonged expiration and expiratory whee throughout the both lung fields. Tactile fremitus and vocal Resonance are bilateral comparable.

On extending my examination trachea is central in position with tracheal Tug reduced crico-sternal distance & prolonged forced expiratory time. Apex beat w difficult to localize. Fingers are not clubbed & there is no evidence of peripheral central cyanosis. There are no flapping tremors, palmarerythema or bounding puls Venous pressure is not elevated & there is no pitting ankle or sacral edema & pulmon component of 2nd heat sound is loud.

My Clinical Diagnosis based on these findings is Chronic Obstructive Pulmon Disease with predominant Bronchitis and with evidence of pulmonary hypertension)

Viva questions:

- **What investigation would you like to do?**
  1. Laboratory
     A. Full blood count: anaemia of chronic disease/polycythaemia.
     B. Raised inflammatory markers if co-existent infection, low albu (due to chronic disease).
     C. Arterial blood gases: hypoxaemia and/or hypercapnia
     D. Sputum culture.
     E. Alpha 1 antitrypsin levels if patient is young, minimal smok history or positive family history.

2. Radiological

   A. CXR: Increased radiolucency of lungs, Flattened diaphragm. Tubular heart.

   B. Computed tomography: Highly sensitive and specific. High-resolution CT (HRCT) used to grade and classify emphysema.

3. Functional

   A. Lung function studies: Airflow obstruction with FEV_1/FVC ratio <0.70. FEV1 < 80% of predicted. Minimum reversibility with broncodilator. Reduce diffusion capacity.

4. Others;

   A. BMI

   B. ECG.

   C. Echocardiogram (to evaluate possible pulmonary hypertension).

- **What treatments are available?**

1. Non-pharmacological:

   A. Smoking cessation is the only treatment shown to alter disease course. Suggestions for relapse prevention.

   B. Pulmonary rehabilitation.

   C. Optimize nutrition.

   D. Vaccination: Influenza vaccination should be offered to all patients. And Pneumococcal vaccination in patient > 65 years or 2 exacerbations per year.

2. Pharmalogical treatment.

   A. Long-acting beta 2 agonists.

   B. Long-acting anticholinergics.

   C. Inhaled corticosteroids.

   D. Oral corticosteroids- in exacerbations

   E. Combined treatment with inhaled bronchodilator and corticosteroid

   F. Theophylline

   G. Long-term oxygen therapy (LTOT): Indicated in patients with $PaO2<7.3kPa$ or $paO\ 2 <8.0kPa$ in presence of secondary polycythaemia, nocturnal hypoxaemia, cor pulmonale or pulmonary hypertension. Indicated15 hours a day.

H. Non-invasive ventilation; Considered in patients with chronic type II respiratory failure despite adequate treatment.

- **Describe the surgical options in patients with COPD Lung volume reduction surgery**

1. Lung volume reduction surgery
2. Bullectomy
3. Lung transplant

- **What causes an acute exacerbation of COPD?**

1. Infection (60 %)
2. Environmental pollution (10 %).
3. Unknown aetiology (30 %).

- **How are infective exacerbations of COPD treated?**
  - Controlled oxygen therapy to achieve arterial oxygen saturations of 88-92 %.
  - Nebulized beta 2-agonists and anticholinergics.
  - Systemic corticosteroids
  - Antibiotics
  - Aminophylline

4.5: Explanations of Interstitial lung disease:

Middle age lady of normal built sitting on bed with Respiratory distress having respiratory rate of 24 breaths/min. Patient is cyanosed centrally &peripherally.

On examination of back of chest, normal in shape with no scar mark. Chest movements are bilaterally reduced but equal. Chest expansion is less than 3cm.Percussion note is resonant throughout the lung fields. On Auscultation. There are fine end inspiratory crackles which are not changing character with cough. Vocal Resonance & tactilefremitus are normal bilaterally and comparable.

On extending my examination tracheas is central in position with no tracheal tug & normal crico-sternal distance. Fingers are clubbed; venous pressure is elevated & there is pitting ankle edema. Pulmonary component of 2nd heart sound is loud. However there are no stigmata of Rheumatogical diseases. Patientis having thin papery skin, Bruises on forearm, moon like face & increase supra clavicular fat pads and purpuric strias on abdominal skin.

My Clinical Diagnosis is interstitial lung disease with pulmonary hypertension and patient must be on steroid as evidence by cushinoid appearance.

Viva questions:

How would you like to investigate the case?

- Laboratory
  - Arterial blood gas (hypoxaemia and hypocapnia)
  - FBC (eosinophilia)
  - Precipitins (EAA).
  - Autoimmune screening: Anti-nuclear factor, rheumatoid factor (RA), anti-centromere antibody (limited systemic sclerosis), anti-Scl 70 (diffuse systemic sclerosis), anti-Jo 1 (DM).
  - Serum immunoglobulins (bronchiectasis, IgE for ABPA) and serum
  - ACE activity (sarcoidosis).
  - CK (associated myositis).
- Pulmonary function testing with diffusion capacity (DLCO): Generally restrictive defect (FEV 1/FVC > 70 %) with reduced diffusion capacity (DLCO). 6-minute walk test (6MWT) cam help too.
- Radiological
  - CXR: Reticulonodular shadowing in affected areas which advances with disease progression.
  - HRCT: Assesses extent, pattern (reticular or nodular) and distribution (upper or lower zones) of disease.
- Others
  - Echocardiogram (to assess pulmonary artery pressure).
  - Bronchoscopy (with BAL)
  - Lung biopsy – transbronchial, open or video assisted thoracoscopic surgery [VATS])

What are the main treatments available for pulmonary fibrosis?

Treatment options include the following:

- Corticosteroids
- Immunosuppressives (cyclophosphamide, azathioprine, methotrexate and cyclosporine).
- Anti-oxidant (N-acetylcysteine in conjunction with corticosteroids and azathioprine).
- Supplemental oxygen (PaO 2 <7.3kPa or <8.0kPa with cor pulmonale).

- Lung transplantation (60 %1-year survival).

In addition to glucocorticoids and immunosuppressives, are you aware of any other potential treatments?

Agents acting as anti-fibrotics have been suggested for use in the management of pulmonary fibrosis.

4.6: Scheme for Examination of front of the chest

FRONT OF CHEST

(Mostly for apical lung collapse or mass)

- Introduction
- Consent
- Position (supine and arms by side and flexed at elbows to ease examination of axilla.
- Exposure (shirt off)
- Head to toe -- Deep observational look to get any hint.
- Calculate Respiratory rate & Observe Chest shape, movement & scar mark.
- Ask for pain on front of chest.
- Palpate for tenderness.
- Check chest movements at apices & below nipples.
- Skip chest expansion till end when you will sit your patient.
- Tracheal position, Tug, crico-sternal distance.
- Apex beat localization.
- Tactile Fremitus – at apices, rest of lung field and at axillae bilaterally.
- Percussion – apices, clavicle, rest of lung field and axillae bilaterally.
- Auscultation on same areas
- Vocal Resonance on same areas
- Ask patient to sit & measure Chest expansion with measuring Tape.

Relevant Examination:

Ask the patient to Lie Down.

- Palpate apex beat. **(Preferably do it when patient was lying initially to make** more comfortable for patient avoiding repeated sitting and lying)

Ask the patient to stretch hands for

- Flapping tremors.
- Check clubbing.
- Look for palmar erythema.
- Check Tenderness at wrist (Hypertrophic osteoarthropathy) & sensory loss at C8 T1 (brachial pluxes involvement) if you found clubbing & suspecting apical tumor.
- Check for bounding pulses.
- Look at eyes for Jaundice, Pallor and meiosis (Horner's Syndrome if suspecting apical mass).
- Check Jugular venous pressure (JVP)
- If JVP raised than check for hepatomegaly & ascites.
- Pedal edema.
- Auscultate pulmonary area for loud P2.
- Check forced expiratory Time if Suspecting COPD. (Keep it to end).
- Ask patient to sit & Check lymph nodes (cervical & axillary).
- Check Bony Tenderness at sternum or vertebral bodies where you want.

4.7: Explanation of lung mass/ collapse

Old age emaciated man sitting on bed with branula on Right forearm, in Respiratory distress with R/R of 24 breaths/min. There is no evidence of central or peripheral cyanosis.

On examination of front of the Chest, Chest is slightly depressed at right Supra-memory region. However there is no scar mark. Trachea is deviated Towards Right side. However apex beat is not displaced. Chest movements are reduced on right supra-memory region& chest expansion is less than 3cm.Percussion note is dull in the same area. Breath sounds are diminished along with reduced tactilefremitus & vocal Resonance in same area. (Collapse with obstructed bronchus or mass lesion).

OR

There is bronchial breathing along with increased tactile fremitus and vocal Resonance. (Collapse with patent bronchus)

Fingers are clubbed with signs of hypertrophic osteo-arthropathy. There is wasting of small muscles of Right hand along with reduced sensation in C_8-T_1 dermatomes. There is no evidence of Hornors syndrome. There is palpable supraclavicular & cervical lymphadenopathy, largest measuring 2x1 cm. firm in consistency, non-tender, mobile & not tethered to skin or underlying structures. There no overlying draining sinus. Venous pressure is not elevated there is no pitting ank edema.

My clinical Diagnosis is Right Pancoast syndrome due to Right apical carcinoma of lung

Viva questions:

How you investgate?

Laboratory

- BBloods: FBC (anaemia), LFTs (may be elevated with metastatic diseas in the liver; raised alkaline phosphatase (ALP) often secondary to bony metastases), U&Es (SIADH), bone profile (raised calcium may be secondary to bone metastases or tumour secretion of PTH-related peptid
- SSputum cytology: positive in 20–25 %patients with lung cancer.
- PPleural aspiration: 50–100mL detects 65 %malignant effusions, further 30 %of samples positive on second aspiration.

Radiological

- CCXR: spiculated, non-calcified lesions, collapse, consolidation, pleural effusion, rib destruction, intrathoracic metastases, mediastinal or paratracheal enlargement due to lymphadenopathy.
- CCT chest and abdomen with contrast: To evaluate size and stage of tumour, lymph node involvement and metastatic disease.
- PPositron emission tomography (PET)-CT scan: most sensitive techniq for detecting extracranial metastatic disease.
- TTechnetium-labelled bone scan for metastasis
- MMR imaging (MRI): More effective at evaluating spine and brain metastases.
- EEndoscopic ultrasound: role in predicting the resectability of a tumour and identifying and sampling nodal disease.

- LLung function testing: To assess suitability for surgical resection and radical radiotherapy.
- HHistological sampling by
  - PPercutaneous CT-guided biopsy – for peripherally located lung lesio and bony metastases

- UUltrasound-guided biopsy/aspiration– of peripheral lymph nodes, liver metastases
- Fflexible bronchoscopy and BAL to assess position within bronchial tree/BAL for cytology/Endobronchial biopsy or brushings
- VVAT biopsy

Describe the histological classification of lung cancer

- Nnon-small-cell lung cancer (NSCLC) ; 75–80 %of lung cancer. Squamous cell > adenocarcinoma > alveolar cell > large cell.
- Ssmall-cell lung cancer (SCLC) 20–25 %of lung cancer. Rapidly proliferating tumour with early dissemination.

Name the common sites of lung cancer metastases

- Liver
- Adrenal glands
- Bone– osteolytic appearance, most commonly in vertebral bodies
- Brain.

What are the most common paraneoplastic syndromes that affect lung cancer patients?

Musculoskeletal

- Hypertrophic pulmonary osteoarthropathy
- Polymyositis.

Neurological

- Lambert–Eaton myasthenic
- Proximal myopathy.
- Peripheral neuropathy.
- Cerebellar syndrome.

Endocrine

- Hypercalcaemia:
  - Tumour secretion of parathyroid hormone (PTH)-related peptide, calcitriol and other cytokines.
- Syndrome of inappropriate anti-diuretic hormone (SIADH)
- Ectopic adrenocorticotropic hormone (ACTH) secretion
- Cushing's syndrome and pigmentation.

CS CamScanner

  - HCG secreting tumour: gynaecomastia.
- Dermatological
  - Thromboplebitis migrans
  - Acanthosis nigrans
  - Eythema gyratum repens
  - Dermatomyositis.

Describe the management of patients with NSCLC

Surgery: Surgical resection in the form of lobectomy or pneumonectomy offers the best long-term survival for patients with lung cancer. Considered in patients with stage I and II disease and considered on an individual basis in patients with stage III A disease. Patients need to be carefully selected based on adequate lung function and comorbidities.

Radical radiotherapy: CHART (continuous hyperfractionated accelerated radiotherapy) is considered in patients with stage I, II or III disease who are inoperable with a good performance status.

Chemotherapy: Offered to patients with stage III or IV cancer with a good performance status (WHO score 0–1) usually a platinum-based drug (carboplati cisplatin) combined with a 3rd-generation drug (docetaxel, gemcitabine, paclitaxel or vinorelbine).

Combination of chemotherapy and radiotherapy provides a survival advantage in patients with stage III disease.

Describe the management of small cell lung cancer?

Combination treatment with radiotherapy and platinum-based chemotherapy. Prophylactic cranial irradiation considered in patients who respond to treatment-shown to reduce the incidence of brain metastasis.

Chapter – 5

Miscellaneous

5.1: Scheme for General physical examination

Command: Perform General physical examination and do relevant

- Introduction
- Consent and assess speech by asking name
- Exposure (shirt off if male)
- Position
- Take a look from foot end and Try to get hints as much as possible
- Check Temperature
- Examine Pulse —
    - Rate
    - Rhythm
    - Volume
    - Character
    - Radio-radial delay
    - Radio-femoral delay
- Measure blood pressure if you think it's relevant otherwise you can do it at end if time allows.
- Check Respiratory rate.

➤ Ask to stretch hands in front of you.

- Flapping Tremors
- Fine Tremors
- Clubbing at level of hands

➤ Any other nail or skin changes on Dorsum of hand.

- Leakonychia
- Koilonychia

- Splinter heamorrhoges
- Beu's lines
- Periungual Telengiectesia
- Skin Texture
- Muscle wasting

➢ Palmer surface
  - Erythema
  - Pallor
  - Dup. contracture
  - Pigmented creases
  - Swollen/Deformed Joints
  - Osler/Heberden/Bouchered nodules.

➢ Forearm:
  - IV lines
  - Bruises / Petechia
  - Tattoos
  - Rheumatoid nodules/epitrochlear/lymph nodes.

➢ Head
  - Alopacia

➢ Eyes
  - Xanthelasmas
  - Corneal arcus
  - Pallor
  - Jaundice

➢ Face
  - malar rash
  - Beak nose
  - Parotid enlargement

➢ Oral cavity:

- Gum Hyperplasia
- Aphthous ulcers
- Lichen planus
- Petechia/Bleeding spots
- Congested Throat
- Glossitis

➢ Neck

- Jugular venous pressure (Hepato-jugular Reflex)
- Thyroid
- Lymh nodes (cervical and axillary)
- Acanthosis nigricans

➢ Chest

- Spider Navi
- Sternal Tenderness
- Gynecomastia

➢ Inguinal lymph nodes

➢ Pedal edema

Relevant Examnation:

- Abdomen (If palpable lymph nodes)
    - a. Hepatomegaly
    - b. Splenomegaly
    - c. Ascites
- Precordium examination (in anemia)

Chest Auscultation: To rule out pulmonary tuberculosis (if there is palpable Lymph Nodes)

Position Sense: if Anemia (subacute combined degeneration)

.2: Examination scheme for hands and do relevant (rheumatology)

Command: Examine Hands & Do Relevant

- Introduction

- Consent
- Position (place both hands on pillow to examine)
- Ask for any pain in hands?
- Exposure (till elbows)
- Observational look from foot end from head to Toe (to see any walk aids/wrist splint/ facial pigmentation/rash/skin tightening/alopecia/hair line

Inspection:

- Nails changes:
    - Clubbing
    - Pitting
    - Nail fold infarct
    - Splinter hemorrhages
    - Telangiectasia
- Dorsum of hands:
    - Skin Texture/Rash (dermatomysitis/scleroderma)
    - Swollen Joint – pattern (symmetrical- RA, asymterical – Gout/OA/psori arthritis)
    - Muscle wasting
    - Nodules around joints (heberden/buchards nodules)
    - Spindling of fingers/deformities(swan neck/boutonniere/Z-deformity deviation)
- Palm:
    - Pallor
    - Erythema
    - Digital ulcers.
    - Crease Pigmentation
    - Osler/Haberden nodules
    - Swollen / Deformed Joints.
    - Fasciculation
    - Thenar /hypothenar wasting

- Wrist/elbow Deformity
- Scar for carpal tunnel decompression
- Psoriatic Plaques At elbow.

Palpation after asking about pain in hands

- Skin Temperature — compare with normal
- Joint. feel DIP/PIP/MCP joints for
    - Tenderness
    - Swelling/effusion
    - Nodules
- Dup.Contracture
- Dislocation of ulner styloid process

Movements:

- Passive & Active movements around all joints & assess power.
- Squeeze my fingers/open and spread fingers apart
- Prayer sign
- Reverse prayer sign

➤ Functional assessment

- Button and unbutton shirt
- Pick up coin

➤ Pin-prick Sensation along Ulnar/Median/Radial nerve

➤ Phalen Test

➤ Tinel Test

➤ Ask to put hands on shoulder and examine foramn for

- Rheumatoid nodules
- Olecranon bursa
- Psoriatic plaques at elbow
- Epitrochlear lymph nodes

➤ Palpate ear lobe for gouty tophi

- Hair line for psoriasis

Relevant examination:

- Face (cushingnoid, beak nose, malar rash….)
- Eyes (anemia, jaundice, xanthelasmas)
- Other Joints if Joints are involved.
- Spinal tenderness
- Proximal muscle weekness
- Precordium (endocarditis, rheumatic AR….)
- Chest (lung fibrosis/ tuberculosis/sarcoid….)
- Abdomen (hepatosplenomegaly…)
- Lymph nodes

(Do relevant examination according to you findings).

Explanation:

Must comment on:

- Active/inactive inflammation
- Functional status
- Other connective tissue disease i.e SLE/Systemic sclerosis / dermatomysitis / polymyositis
- Systemic complications

There is symmetrical deforming arthropathy in small joints of hands involving MCP and PIP joints and sparing DIP joits. There is spindling of fingers due to soft tissue swelling around MCP and PIP joints. There is palmar sublaxation at MCP joints and ulner deviation of fingers. The deformities involved swan neck, boutonniere and Z-deformity of thumb.

There are nodules on extensor surface of elbows. There is palmar erythema and wasting if small muscles of hands. There are nail fold infarcts and vasculitic skin lesions. The skin is thin and atrophic with purpura on hands and forearm. There is no evidence of entrapement neuropathy. The affected joints are warm, erythematous and tender. Patient is unable to unbutton her/his clothes.

The diagnosis is rheumatoid arthritis. There is evidence of active inflammation and associated disability. No signs of other C.T diseases. I would like to examine for extra-articular manifestation.

Viva questions:

How would you like to investigate?

- Inflammatory markers: CRP, ESR – both elevated.
- FBC that may show:
  - Normochromic normocytic anaemia of chronic disease
  - Low WBC in Felty's syndrome.
- Rheumatoid factor
- Anti-CCP (cyclic citrullinated peptide) antibodies
- X-rays: key signs – periarticular osteopenia, erosions typically at the margins of the small joints, joint space narrowing, deformity.
- Power Doppler ultrasound or MRI to look for active synovitis.
- CXR: pulmonary fibrosis or pulmonary nodules.
- Lung function tests: Restrictive defect: d FVC, i FEV 1/FVC ratio, d TLCO due to pulmonary fibrosis.
- HRCT chest: non-specific interstitial pneumonitis (NSIP) (commonest) but usual interstital pneumonitis (UIP), organizing pneumonia and bronchiolitis all occur.

How would you manage?

- Exercise and physiotherapy
- NSAIDs, and COX-2 inhibitors
- DMARDs:
  - Methotrexate
  - Sulphasalazine
  - Leflunomide
  - Hydroxychloroquine (antimalarial).
- Biologic therapies
  - TNF blockade:
    - Infliximab, adalimumab and golimumab (anti-TNF monoclonal antibodies)
    - Etanercept (soluble TNF receptor antagonist)
    - Certiluzumab pegol (pegylated Fab construct)
  - Newer biologic therapies:

- Rituximab (anti-CD20 monoclonal antibody which depletes B cells)
- Abatacept (CTLA4-Ig which blocks co-stimulation of T cells)
- Tociluzimab (anti-IL-6 receptor monoclonal antibody).

What are the side effects of methotrexate?

- Hepatitis, alveolitis, stomatitis.
- Toxicity due to overdose: marrow suppression/pancytopenia leading t opportunistic infections (e.g. PCP).
- Methotrexate is given once weekly and the commonest cause of toxicity is bei incorrectly prescribed or being taken as daily dose.

What is the treatment for methotrexate toxicity?

Folinic acid (works quicker than folic acid).

How do you manage a patient on anti-TNF treatment who presents to A&E with minor infection such as cellulitis?

- Admit the patient
- Stop anti-TNF Rx
- Intravenous antibiotics
- Seek specialist advice.

How do you distinguish rheumatoid arthritis (RA) from osteoarthritis (OA) on x ray?

- RA: juxta-articular osteopenia, erosions, symmetry, deformity.
- OA: sclerosis, osteophytes.
- Both: bone cysts, joint space narrowing.

How do you distinguish active from inactive arthritis?

Symptoms (increased pain, fatigue), swelling, joint tenderness, warmth of joints raised ESR. Disease activity may be masked in a patient taking NSAIDs.

Scleroderma case: explantion

This patient have sclerotic skin over the face and fingers that appears smooth shiny and tight. There is loss of facial wrinkes, perioral puckering with restrictive mouth opening and the nose appeared pinched. There are telengiectesias on face. On examination there is sclerodactyly with flexion deformities. There are dilated nail fold capillieries with dystrophic nails. There is atrophy of soft tissue particularly in finger pulps with digital ulceration and gangrene. There are palpable nodules of calcinosis. Diagnosis is systemic sclerosis.

I would like to examine lungs- ILD/ precordium- pul.HTN/ GI- primary biliary cirrhosis/ signs of renal failure.

Viva questions:

How would you like to investigate?

CBC- anemia

Urea and elctrolytes

LFTs

Autoimmune screening

- Positive ANA in 90 %of patients; anti-Scl-70 (anti-topoisomerase) in 40 %of patients with dcSSc and anti-centromere antibodies in 80 %of patients with lcSSc.

Nailfold capillaroscopy: enlarged capillaries, microhaemorrhages, dropouts.

Lung function tests: decreased transfer factor.

HRCT: features of ILD.

Echo for RVH and increased pulmonary pressures.

Radiographs may demonstrate acro-osteolysis of terminal phalanges, subcutaneous calcinosis (in CREST variant); erosive arthritis is rare.

How would you manage?

Analgesia and physiotherapy

Raynauds can be treated with –

- Smoking cessation
- Warming
- Vasodilators

GI symptoms can be treated with

- Nutrional supports
- Antibiotics for bacterial overgrowth
- Proton pumo inhibitors
- Prokinetics

Renal complications – ACEI for HTN and proteinuria

Respiratory complications -

- vasodilators for pul HTN
- Antifibrotic therapy for lung fibrosis

Explanation of dermatomypsitis:

There is symmetrical heliotrope rash around the eyes. There are flat topped violaceous, scaly papules and plaques over the knuckes/elbows and knees (Gottran papules). There are dilated nail fold capilleries with periungual telengiectesias. There is erythamtous rash in sun exposed distribution- extonser surfaces of arms, V- shape distribution on anterior neck, upper chest and back. There is proximal muscle weekness and tenderness. I couldn't appreciate any small or large joint involvement or features of other C.T diseases.

Most likely diagnosis is dermatmyositis. I would like to do complete clinical examination to rule out internal malignancy.

Viva questions:

How would you investigate?

- CBC and raised inflammatory markers
- CK levels
- LFTS/RFTS
- Blood glucose and HBA1c
- Thyroid function tests
- Autoimmune screen- eNA profule
- CXR and HRCT to see lung involvement
- Echo to see cardiomyopathy
- EMG- myopathic pattern
- Muscle MRI
- Muscle biopsy

How would you manage?

Physiotherapy and analgesia

Skin changes- avoid sun exposure and sun screen and hydroxychroquine

Muscle – high dose steroids 1mg/kg/day followed by DMARDs (azathioprine, methotrexate) in refractory cases, ciclosporin, cyclophophosphamide or IV immunoglobulin.

ich patients are at increased risk of a statin-induced myopathy?

There is an eightfold increase in myopathy in patients taking statins, increasing to a 42-old risk if a fibrate is co-administered. The risk is dose related, but is higher in the lderly, diabetics, hypothyroidism, or concurrent renal or liver disease. Drugs such as iclosporin, proton pump inhibitors, calcium-channel blockers, selective serotonin euptake inhibitors (SSRIs), and grapefruit juice all increase the risk.

.3: Examination scheme in Diabetes Mellitus

- Cachectic / Obese
- Skin Pigmentation
- Hands
    - Onychomycosis.
    - Dupuytren's Contracture
    - Pallor
    - BSL check Prick mark.
- Pulse with Radio-radial / radio- Femoral Delay & Peripheral Pulses.
- Blood pressure with postural Drop.
- Acanthosis nigricans
- Eyes:
    - Xanthelasmas/peri-orbital puffiness
    - Ocular palsy
    - Corneal arcus
    - Cataract
    - Fundus examination
- Oral
    - Thrush
    - Poor Dental Hygiene.
- Neck
    - Carotids
- Skin

  - Insulin injection marks
  - Lipo-dystrophy
  - Vitilogo, hair loss
- Proximal myopathy.
- Joint Deformity (Charcot joint)
- Peripheral Sensation
  - Pin prick
  - Vibration
  - Position
- Foot
  - Ulcer
  - Gangrene
  - Calluses.

Relevant

- Planter response bilaterally
- Precordium – third heart sound
- Cushinoid features

Ask for Blood sugar level Chart, Urine for protein & Ketones.

5.4: Examination scheme in Cushing disease

- General Look

Hands

- Clubbing
- Fungal infection
- Palmer erythema
- Deformed/Swollen joints (Rheumatoid Arthritis)

Arm

- Bruises
- Thin atrophic skin

CS CamScanner

- Proximal myopathy
- Blood pressure

Neck and Axillae

- Acanthosis nigricans
- Supra clavicular/ Inter-scapular fat pads.
- Telangiectasia

Face

- Moon face
- Cataract /Hemianopia (Pituitary tumor)
- Acne
- Hirsutism
- Oral Thrush
- Malar Rash
- Precordium

Chest

- Lung Carcinoma
- Chronic obstructive pulmonary disease
- Interstitial lung disease

Abdomen.

- Purpuric striae
- Obesity
- Scar
- Mass
- Lipodystrophy (Insulin)

enitalia -Virilisation

ack

- Vertebral Tenderness
- Kyphosis

Explanation:

The patient has cushingoid facies with centripetal obesity. There ar interscapular and suprascapular fat pads. There is acne vulgaris on the face and the skin is thin and atrophic with purpuric and petechial rash consistant with bruising. The is evidence of hirsutism on face and onycholysis on finger nails. There are violaceou striae on abdominal skin. There is evidence of proximal myopathy. There is a adrenalectomy scar. Most likely diagnosis is Cushing syndrome.

Viva questions:

How would you investigate?

Confirmation of hypercortisolism

- Overnight dexamethasone suppression test (1mg dexamethasone at midnight an check 9.00am serum cortisol. This test is considered abnormal if the cortisol lev is > 50nmol/L).
- 3 ×24-hour urine free cortisol measurements.

If positive proceed to Identification of the cause of Cushing's syndrome

- Plasma ACTH level: ACTH levels should be undetectable in adrenal Cushing but will be inappropriately 'normal' or elevated in pituitary (Cushing's disea or ectopic Cushing's syndrome.
- High dose dexamethasone suppression test (give 2mg dexamethasone 6-hour for 48 hours and measure a baseline cortisol and a 48-hours cortisol. Corti levels should fall by > 50 %of baseline in pituitary Cushing's, but not adrenal o ectopic.
- Corticotropin releasing hormone (CRH) test will cause a rise in cortisol in 90% individuals with pituitary Cushing's but should not cause a rise in indiv with adrenal or ectopic Cushing's.

Radiological localization (only proceed after biochemical confirmation)

- Adrenal CT: incidentalomas can cause problems in diagnosis.
- MRI pituitary: will identify 50-80 %of ACTH secreting microadenomas.
- Inferior petrosal sinus sampling if still confusion on cause.
- Consider HRCT chest to look for ectopic source of ACTH.

What are treatment options?

Most patients will be started on metyrapone whilst awaiting definitive treatment.

Treatment of pituitary Cushing's:

- Transphenoidal resection

- Pituitary radiotherapy
- Bilateral adrenalectomy (rarely done)

Treatment of adrenal Cushing's:

- Adrenalectomy
- Ketoconazole
- Mitotane (if Cushing's caused by adrenal carcinoma)

What are the causes of hypercortisolism?

- Exogenous steroids
- Pituitary adenoma (Cushing's disease)
- Adrenal adenoma/hyperplasia
- Adrenal carcinoma
- Ectopic ACTH (e.g. small cell carcinoma of the lungs).

5.5: Examination scheme in acromegaly

Voice:

- Husky (note while taking consent)

Hands:

- Large warm, sweaty
- Doughy feelings
- Spade like fingers
- Thick dorsal skin
- Carpal tunnel syndrome signs
- Clubbing
- Needle prick marks for DM

Arms:

- Proximal myopathy

Neck:

- Goiter
- A [illegible]

Face:

- Large course facies
- Prominent Forehead wrinkling
- Prominent supra orbital ridge
- Baggy eyelids
- Large protruding lower jaw (prognathinism)
- Large lips, nose, ears
- Maloccluded widely apart teeth
- Large tongue
- Enlarged skull bones

Eyes:

- Bitemporal hemianopia (field of vision)
- Papilledema
- Optic atrophy

Skin:

- Thick sweaty greasy pigmented

Chest:

- Gynecomastia
- Galactorrhea

Precordium:

- Cardiomyopathy

Abdomen:

- Visceromegaly

Lower limbs:

- Enlarged feet
- Pedal edema
- Knee arthroplasty

Spine:

- Kyphosis

Joints:

- Arthritis

Explanations of acromegaly:

The patient have large spade like hands with sweaty palms. Skin over the dorsum of hands is thickened. Face has prominent supraorbital ridges, large nose and lips and proganthanism. There is interdental separation and tongue is enlarged (macroglossia). There is evidence of hypertrichosis, gynecomastia and acanthosis nigricans over neck.

Likely diagnosis is acromegaly. I would like to measure BP, look at the chart and dip the urine for glycosuria. Offer to palpate the abdomen (for colonic masses and hepatosplenomegaly) if not done already.

Viva questioions

How would you investigate?

- OGTT- Failure of growth hormone (GH) to suppress to <2nmol/L following an OGTT. (Random GH measurements are not diagnostic because of the episodic secretion of GH, its short half-life, and the overlap between GH concentration in individuals with and without acromegaly.)
- IGF 1: used to monitor disease progression.
- MRI pituitary (no need for lateral skull x-rays!).
- Formal Goldman or Humphrey visual fields.
- Test anterior pituitary function
    - Check T 4 (TSH unhelpful in pituitary disease).
    - LH, FSH and testosterone/oestradiol (ensure FSH/LH appropriate).
    - Short Synacthen test, ACTH and cortisol levels (ensure ACTH appropriate).

How would you treat?

- Surgery: Surgery is the treatment of choice, and trans-sphenoidal surgery is usually the best option.
- Radiotherapy can be used as an adjunct to surgery and in those unfit for an operation, but may take many years to have full effect.
- Drug therapy is often used preoperatively and can also be a useful adjunct to surgery.

- Somatostatin analogues (e.g. octreotide) reduce GH secretion but do not significantly reduce tumour size. Valuable role in reducing hypersecretion post surgery.
- Dopamine agonists (e.g. bromocripine) only effective in <20 %of cases.
- Pegvisomont, a competitive GH antagonist, normalizes IGF1 levels and rapidly improves active symptoms. Although well tolerated, it is expensive and has to be given subcutaneously. It does not reduce tumour size.

What are the complications of acromegaly?

- Diabetes
- Hypertension
- Cardiomyopathy
- OSA
- Colonic polyps/bowel cancer
- OA
- Hypercalciuria.

What signs and symptoms may indicate active disease?

- Sweating
- Skin tags
- Hypertension
- Peripheral oedema.

What are the causes of macroglossia?

- Acromegaly
- Amyloidosis
- Hypothyroidism
- Down's syndrome.

What are the complications of pituitary surgery?

- Diabetes insipidus (may be transient)
- SIADH (transient)
- Hypopituitarism
- CSF leak

- Intracranial bleeding.

5.6: Examine thyroid and do relevant examination

Thyroid gland

Inspection

Swallow test, tongue protrusion test

Palpation

Size, consistency, surface, tenderness, temperature, overlying skin fixation

Percussion

At sternum to check retro sternal extension

Auscultations – thyroid bruit

Neck

- Lymph nodes - cervical and axillary
- Carotid pulse

Hands - tremors, sweating, warm, Palmer erythema, clubbing (thyroid acropathy), Carpal tunnes signs (tinnel/phelen test)

Pulse -- irregular, good volume

Blood pressure— may be high

Arms – proximal myopathy

Eye

- Exophthalmos
- Lid lag
- Lid retraction
- Ophthalmoloplegia
- Diplopia
- Chemosis
- Redness

Face - anxious, restless, staring look

Skin --pretibial myxedema, vitiligo

Precordium -- flow murmur

Brisk or delayed reflexes

Relevant Examination:

- Joints (RA)
- Rash (SLE)
- Dry eyes
- Finger prick marks (Diabetes mellitus)
- Skin crease pigmentation (Addison's)
- Phalen and tinnel signs for carpal tunnel syndrome
- Fundoscopy (Diabetes mellitus)

Additional features can be seen in hypothyroidism

- Slow mentation
- Speech- hoarse coarse husky voice
- Depressed, puffy face
- Loss of eyebrows outer third
- Xanthelasmas

Relevant for hypothyroid can include:

- Look for position vibration sense (vitamin B 12 deficiency)
- Cerebellar ataxia
- Joints (RA)
- Rash (SLE)
- Dry eyes
- Finger prick marks (DM)
- Skin crease pigmentation (Addison's)
- Phalen and tinnel signs for carpal tunnel syndrome
- Fundoscopy (DM)

Explanation hyperthyroidism:

The patient is thin, anxious and restless. The hands are sweaty with palmer erythema and fine tremors on outstretched hands. Pulse is tachycardiac and irregular. There is lid lag and lid retraction. There is evidence of proximal myopathy. There is no palpable goiter and no evidence of previous thyroidectomy. I couldn't appreciate any features of graves disease (ophthalmoplegia/acropathy/pretibial myxedema).

Likely diagnosis is hypothyroidism with atrial fibrillation.

xplanation hypothyroidism:

Patient have elevated body mass index. There is generalized slowness of movements and speech. Speech is hoarse. Skin is course, dry with cool peripheries. Pulse s regular and bradycardiac. There is periorbital swelling and generalized non pitting dema. Thyroid gland is palpable and smoothly enlarged and non tender with no vidence of retrosternal extension and with no bruit. No palpable cervical ymphadenopathy. There is no evidence of proximal myopathy, lid lag/retraction or any car for previous thyroid surgery. No features of graves disease.

Likely diagnosis is hypothyroidism with goiter.

iva questions:

ow would you investigate?

- TSH, T 3, T 4
- Thyroid auto-antibodies (and anti-TSH receptor antibodies, TPO antibodies)
- Thyroid uptake scan
- Thyroid ultrasound is helpful for detecting impalpable nodules.
- CT thorax and neck are useful for large goitres to assess for compression.
- Fine-needle aspiration

ow would you treat hyperthyroidism?

rugs:

- Carbimazole and propylthiouracil
- Given by 'titration' or 'block and replace'. Typically in the titration regime, an initial dose of carbimazole 30–40mg is tapered down over 4–8 weeks to a maintenance dose of 5–10mg. In the block and replace regime typically 40mg of carbimazole is used initially and 100mg of levothyroxine added when the free T 4 is in the normal range (usually after 4 weeks) to prevent hypothyroidism.
- Treatment course for both regimes is typically 18 months.
- Warn about rash (1:10) and risk of agranulocytosis (1:200; should mouth ulcers develop, stop carbimazole immediately and check neutrophil count).

dioiodine:

- Likely to result in long-term hypothyroidism.

- May exacerbate eye disease.

Surgery:

- Effective.
- May result in hoarse voice (following damage to laryngeal nerves).
- Results in long-term hypothyroidism.
- May cause damage to parathyroid glands.

Treatment of symptoms

Non-selective beta-blockers e.g. proponalol (caution in asthma/COPD).

How to treat hypothyroidism?

- Levothyroxine replacement and adjust according to TSH. Caution in the eld Once maintained on a steady dose, annual blood tests are

What are the causes of hypothyroidism?

- Hashimoto's thyroiditis (autoimmune; most common).
- Following thyroid ablation, by surgery or radioiodine.
- Thyroiditis: post-transient hyperthyroid stage.
- Drugs: amiodarone, lithium, interferon, anti-thyroid drugs.

What are the indications for treatment in non-toxic multinodular goitre?

- Compression and obstruction – trachea, oesophagus, venous outflow
- Cosmetic
- Marked intra-thoracic extension

What is the differential diagnosis of a solitary thyroid nodule?

- Thyroid cyst
- Thyroid adenoma
- Thyroid carcinoma
- Metastatic cancer
- Lymphoma
- Sarcoma
- Parathyroid cyst.

Examination scheme in anemia

General physical examination

- Pallor
- Koilonychias
- Leukonychia (GI losses)
- Bruises/ petechia (aplastic anemia)
- Pulse tachycardia with good volume
- Periorbital puffiness
- Angular stomatitis
- Atrophic glossitis
- Chronic pharyngitis
- Lymph nodes
- Jugular venous pressure
- Bony tenderness
- Pedal edema

Precordial examination - heaving apex, flow murmurs

Lungs -- pulmonary edema (high output cardiac failure)

Abdomen - spleen, liver, lymph nodes, per rectal examination for hemorrhoids

Neurological— vibration and position sense (B12 deficeiency)

Proximal myopathy (Vitamin D deficiency)

I would like to ask about dietary habits and menstrual loss in female patient

Chapter – 6

Long Case

6.1: How to take good History

History Taking

- Set your mnemonics for Name, age, gender, occupation, address, date and time of admission, mode of admission, maritalstatus's, past, medica family, Drug, personnel, social, travel history etc& put it on History takin paper in start, so there would be less chances to miss anything.
- Keep Presenting complaints as simple & as minimum as possible (ideal there should be only one presenting complain).
- Describe past history & functional status in start of HOPI, in one or tw lines if it is relevant. e.g "My Patient is normo glycemic, Normotensive known smoker, having recurrent attacks of shortness of breath & productive cough over past 3 years...." Now presented with worsening o Shortness of breath ...."
- Describe ODPARA for each complains.
    - Onset
    - Duration
    - Progression- over time & Severity
        - Aggravating factors
        - Relieving Factors
        - Association

(Describe Functional Status with Progression)

Patients is suffering from shortness of breath for last 1 month, it started graduall & Progressive in nature, initially patient gets Short of breath on climbing stairs, howeve for past 1 week, Patient gets short of breath even on walking few steps on flat. Thi shortness of breath gets worsened by mild exertion & lying flat on bed & patient fe better in leaning forward position. Patient use 2-3 pillow to prop-up himself at night.

(Describe other factors if relevant like use of diuretics, Inhalers etc).

- In Associated Factors, mention positive factors first, then rule out all negative cause (making it easier for you & examiner) So for good history taking you should kno all the possible causes of presenting complain, common to.

For example for shortness of breath:

Cardiovascular causes.

- Ischemia / Infarction
- Arrhythmias
- Valvular Heart diseases
- Heart failure

Respiratory

- Chronic obstructive pulmonary disease
- Asthma
- Interstitial lung disease
- Pulmonary embolism
- Severe pneumonia
- Tuberculosis
- Lung Carcinoma

Anemia

Musk-Skeletal

- Obstructive sleep apnea
- Kypho-Scloliosis
- Myasthenia / GBS.

"There is associated history of Dry cough that get worsened at night, chest tightness & wheezing. Patient also gives history of post-nasal drip, seasonal variation, and frequent skin allergies."

"However there is no associated history of productive cough, Paroxysmal nocturnal dyspnea, palpitations chest pain, fever weight loss, leg swelling or prolonged immobilization, or Travel history or muscle weakness, bird or pet, farm or factory exposure."

- Right now you must have reached some Diagnosis. Now inquire about possible complication, association, Risk factor & etiology (CARE)
- Try to complete your history in 10 minutes, afterward you can ask remaining questions during examination.

"This was just to facilitate you, hopefully you have gotten now the main idea behind history taking. Now practice this scheme & built your own histories relevant to each.

possible complain & Try to memorize them, so you can speak out fluently duri
presentation in exam".

Examination of long case

- Take vitals 1st as they are vital to take:
    - Temperature
    - Pulse
    - Respiratory rate
    - Blood pressure

(Don't forget to take Thermometer back).

- Perform General Physical examination in Detail.
- Perform relevant system Examination thoroughly initially, followed b examination of other systems in detail as Time allows.

6.2: Important History Points for Jaundice

- ODPARA
- Take History Relevant to causes.
- Pre-Hepatic
    - Hemolytic
    - Buddchiaris
    - Congestive Hepatic
- Hepatic
    - Acute viral
    - Chronic viral hepatitis
    - Alcoholic
    - Hemochromatosis
    - Wilson's disease
    - Auto immune hepatitis
    - primary biliary cirrhosis
    - Carcinoma
    - Alfa -1 antitrypsin Deficiency

Post-Hepatic (Obstructive)

- Gallstone
- lymphoma
- carcinoma

CARE
(Complication, Association, RISK factors, ETIOLOGY)

6.3: Important History Points for Abdominal Swelling

- ODPARA
- Rule out causes
  - Decompensated chronic liver disease
  - Congestive cardiac failure
  - Chronic kidney disease/Nephrotic syndrome
  - Malabsorption
  - Tuberculosis
  - SLE
  - Lymphoma
  - Carcinomatosis
  - Meigs Syndrome

CARE (according to relevant cause)

6.4: Important history points for Joint Pain

- ODPARA
- Rule out causes
  - Rheumatoid arthritis
  - Systemic lupus erythamatosis
  - Anti phospholipid syndrome
  - Bachet's syndrome
  - Reactive arthritis
  - Enteropathic arthritis
  - Whipple disease
  - Ankylosing Spondylitis

- Psoriatic arthritis
- Polymyalgia rheumatica
- Rheumatic Fever
- Stills Disease
- Hemochromatosis
- Primary biliary cirrhosis
- Sarcoidosis
- Scleroderma
- Tuberculosis
- Gout
- Osteoarthritis
- Osteomalacia
- CARE
- Functional Status. (GALS Screen)
    - G-Gait
    - Arm-combing
    - L-Leg- stairs
    - S-Spine

6.5: Important History Points for Diarrhea

- ODPARA
- F_2CCV
    - Frequency
    - Foul smell
    - Color
    - Consistency
    - Volume

Rule out causes.

- Celiac disease
- Lactose intolerance
- Inflammatory bowel disease
- Whipples disease
- Osmotic diarrhea

- Secretory diarrhea
- Laxative abuse
- Tuberculosis
- HIV
- Giardiasis
- Primary biliary cirrhosis
- Pancreatitis
- Bacterial overgrowth
- Diabetes mellitus
- Carcinoid
- Thyrotoxicosis
- Pellagra
- CARE

6.6: Important History Points for Fever

- Grade
- Pattern
- Fever free interval
- ODPARA
- Rule out causes
    - Meningitis / Encephalitis
    - Otitis media
    - Sinusitis
    - Eye infection
    - Dental infection
    - Respiratory tract infection
    - Infective Endocarditis
    - Rheumatic fever
    - Hyperthyroidism
    - Diarrhea
    - Cholecystitis
    - Hepatitis

- UTI
- Pyelonephritis
- Osteomyelitis
- Skin infection
- Tuberculosis
- Malignancy
- Lymphoproliferative disorder
- Myeloproliferative disorder
- Aplastic Anemia
- Autoimmune Screen

CARE

6.7: Important History Points for Shortness Of Breath

- ODPARA
- Causes

Respiratory

- Chronic obstructive airway disease
- Asthma
- Interstitial lung disease
- Tuberculosis
- Lung Carcinoma/metastasis
- Severe pneumonia
- Pulmonary embolism
- Sarcoidosis
- Pleural effusion. (Causes)

- CVS
  - Ischemia
  - Heart failure
  - Valvular heart disease
  - Arrhythmias
- Anemia
- Muskulo Skeletal
  - Obstructive sleep apnea

- kypho-Scoliosis
- Pick-wickian syndrome
- GBS/Myasthenia gravis
  - CARE

Modes of onset, duration and progression

Minutes

- Pulmonary thromboembolism
- Pneumothorax
- Acute left ventricular failure
- Asthma
- Inhaled foreign body

Hours to days

- Pneumonia
- Asthma
- Exacerbation of COPD

Weeks to months

- Anemia
- Pleural effusion
- Respiratory neuromuscular disorders

Months to years

- COPD
- Pulmonary fibrosis
- Pulmonary tuberculosis

Severity of breathlessness: Medical Research Council (MRC) classification

Grade 1: Breathless when hurrying on the level or walking up a slight hill

Grade 2: Breathless when walking with people of own age or on level ground

Grade 3: Has to stop because of breathlessness when walking on level ground at own pace

6.8: Important History Points for Cough

- ODPARA
- Rule out causes
  - Respiratory:

(Same as in SOB)

- Heart failure
- Drug induce
  - ACE.I
  - B-Blocker
- CARE

Description of Important histories

6.9: History description of chronic diarrhea

Patient gives history of loose stools for the last one year, gradual in onset and progressive. Initialy 5-6 episodes per day and now 10-12 episodes per day, watery in consistency, yellow color, large in volume, foul smelling, remained same throughout the day and night (even have to go to washroom at night). There is no relationship with any specific kind of food like wheat or milk. And it doesn't improve with fasting. There is history of passing blood with stools, large in amount, mixed with stool contents, 2-3 times per day; however, there is no history of bleeding from any site or bruises or petechias. There is associated history of abdominal pain, colicky in nature, moderate in intensity, generalized, and partially relieved by defecation. There is also associated history of vomiting 4-5 episodes per day, gradual in onset, non-progressive, containing food particles and no blood. Aggravated by taking food. There is associated history of retrosternal burning. No history of epigastric pain, use of NSAIDS or steroids, no history of photophobia, jaundice, oliguria or flank pain.

No associated history of skin rash, joint pain, oral or perineal ulcers, pruritus, jaundice, heat intolerance. No history of past tuberculosis or contact with tuberculosis. No history of travel abroad. No history of facial flushing, wheezing from chest, laxative abuse, diabetes mellitus polyphagia, polyuria, polydipsia, no history of any abdominal surgery. No history of fever, weight loss, anorexia, lumps or bumps. No history of intravenous drug abuse or extramarital affairs.

There is history of palpitations, easy fatigability, blackouts on sudden standing. History of shortness of breathes on exertion However no history of orthopnea, paroxysmal nocturnal dyspnea. Patient has difficulty in climbing stairs and combing hairs. History of gradual abdominal distention and ankle edema, however no periorbital swelling, frothy urine or distended neck veins.

6.10: History description of Joint pain

History of joint pain for the last one year, gradual in onset, started from right knee joint and progressed to other joints including small joints of hands, over the period of few months, without improvement of initially affected joints. Initially pain was mild and he was able to perform daily activities, but now pain is increased in intensity, rendering him to dress independently and walk without support. Pain was more severe in morning and aggravated by activity and partially improved by taking analgesics which patients is unable to recall name. Pain was associated with swelling and redness in joints especially joints of hands. Swelling partially relieved by taking medication. However, now patients are having swelling in —— joints. There is also history of morning stiffness lastin greater than one hour. There is no history of alopecia, photosensitivity, skin rash tightening of skin, bluish discolorations of fingers, and dryness in eyes or proxima muscle weakness. There is also no history of oral or genital ulcers, frothy urine, dysuria pyuria, hematuria, loose stools, blood with stools, back pain. No history of miscarriage unilateral leg swelling, skin darkening, hemoptysis, lumps or bumps, fever, or weigh loss. No history of dry cough or shortness of breath (ILD). No history of periorbita puffiness, ankle swelling or frothy urine (Renal amyloid). No history of weakness on an part of body (Mononeuritis multiplex). No history of palpitations or easy fatigabili (Anemia). Patient complains of thin, dry skin, decrease vision, bruises, puffy face increase weight gain, above pain and difficulty rising from sitting position (Steroi complications)

6.11: History description of Upper GI bleed:

Patient is known case of chronic liver disease was in usual state of health 8 day back when he developed hematemesis, multiple episodes 2-3/hour, containing variabl amount of fresh blood ranging from 2-3 table spoon to 1 cup in amount. Vomitus als contains clotted blood. There is also history of hematochezia, large in amount, multipl episodes, containing both fresh and clotting blood. However there is no history bleeding from any other site. Although patient complains about easy bruisibility. Patien gives history of jaundice for last 1 year that was noticed by his family and investigated be HCV Positive. There is associated history of yellowish discoloration of urine howeve no history of Clay color stools or pruritus. There is associated history of abdomina distension for last 1 year that was gradual in onset and intermittent. Relieved by use Aldactone, recur on drug withdrawal. No associated history of periorbital puffiness ankle swelling, or distended neck veins or diarrhea. Patient give history of multipl hospital admission due to melena, however there is no history of irrelevant talk or altere state of consciousness. Patient gives history of multiple blood transfusion over the las one year Due to melena. Patient gets shave from barber each time with new razo however there is no history of skin piercing, tattoo, alcohol or intravenous drug abuse extramarital affairs, hakeem medication. There is no history of oral ulcer. joint pain alopecia, skin rash or pigmentation, learning difficulties, abnormal limb movements diabetes Mellitus.

6.12: History description of Hemiplegia:

Patient is known hypertensive and diabetic with poor compliance to medication and known smoker. He was in usual state of health 2 days back when he develope weakness in left side of the body while sleeping, it was sudden in onset, simultaneousl affecting left upper and lower limbs and partially improved over the period of two days Initially he was unable to move left limbs completely however now he can move them little bit while lying on bed. It was associated with deviation in angle of mouth, difficulty in speech and swallowing, numbness on the left side of the body. Although there is no history of altered state of consciousness, visual impairment, fecal or urinary incontinence, seizures or tremors. There is also no history of headache Vomiting, neck stiffness, fever, weight loss, lump and bumps or trauma to head. No history of palpitations, orthopnea, paroxysmal nocturnal dyspnea or high blood cholesterol levels. No history of any unilateral leg swelling, any bleeding or clotting disorder, early morning hematuria, learning difficulties or developmental problems. No history of oral ulcer, alopecia, rash, joint pain (recurrent miscarriages in female). No history of cocaine or amphetamine abuse, or use of antiplatelet or anticoagulants, or oral contraceptives. No history of any such past episode.

6.13: History description of Paraplegia:

Patient gave history of weakness of both lower limbs for last 4 months, it was gradual in onset and progressive, involving both lower limbs simultaneously, initially patients was able to walk with support however now he is bed bound for last 3 days. Initially it affected the lower limbs, later on progressed to upper limbs over the period of – days. Now patient is not able to comb, eat and dress him independently. There is history of drooling of saliva, inability to close eyelids, dropping of eyelid, diplopia, difficulty in speech and swallowing. However no history of difficulty in breathing. There is history of numbness and tingling in hands and feet. No history of tendency to fall towards one side or tremors. Fecal or urinary incontinence or altered state of consciousness. No history of diurnal variation or aggravation with any specific food or medication or fasting. No history of preceding diarrhea, sore throats, or vaccination. No history of heat intolerance, palpitations or severe abdominal pain. No history of trauma to spine, pain in backbone, fever, night sweats, weight loss, lumps and bumps, or steroid intake, palpitations, hypercholesteremia, joint pains. No history of headache, vomiting, pallor, numbness in hands and feet. No family history of such weakness or extramarital affairs.

6.14: History description of Shortness of breath:

"My Patient normoglycemic. Normotensive, known smoker 10pack per year, having recurrent attacks of shortness of breath & productive cough over past 3 years…." Now presented with worsening SOB ……… it started gradually & Progressive, initially Patient get Short of breath on climbing stairs, however for past 1 week, "Patient feel short of breath even on walking few steps on flat. This shortness of breath get worsened by mild exertion & lying flat on bed & patient feel better in leaning forward position. Patient use 2-3 pillow to prop-up himself at night. This shortness of breath initially gets better with use of inhaler however now despite using inhaler patient remained short of breath al over the day. Although he is well aware of good inhaler technique. There is associate history of Dry cough that get worsened at night, chest tightness & wheezing. Patient als gives history of post-nasal drip, seasonal variation, and frequent skin allergies. Howeve there is no associated history, Paroxysmal nocturnal dyspnea, palpation chest pain, feve weight loss, leg swelling or prolonged immobilization, or Travel history or musc weakness, bird or pet, farm or factory exposure.

Chapter – 7

Common Causes of Important Clinical Signs

Causes of hypotonia

- LMN lesion (due to any cause)
- Cerebellar lesion (knee jerk may be pendular)
- Dorsal column lesion.
- Polyneuropathy.
- Hypokalemia or hyperkalemia.
- Drug: Any muscle relaxant

Causes of hypertonia

- UMN lesion (spastic, may be clasp knife).
- Extra pyramidal lesion (lead pipe or cogwheel). Conversion disorder {rigidity continues to increase with more and more passive movement).
- Others: Catatonic state, tetanus and strychnine poisoning.

Causes of loss of tendon reflex

- LMN lesion.
- Peripheral neuropathy.
- Dorsal column lesion.
- Hypokalemia or hyperkalemia.

Causes of extensor plantar response

- First year of life (due to lack of myelination of pyramidal tract. Myelination occurs in 6-12 months).
- Sleep.
- UMN lesion.
- Deep coma (due to any cause).
- Postepileptic period (after grand mal seizure).

Causes of distal wasting and weakness

- Myotonic dystrophy

- Distal spinal muscular atrophy
- Inclusion body myositis
- Oculopharyngodistal myopathy
- Walender distal myopathy

Causes of spastic paraplegia

- Spinal cord compression due to any cause
- Demyelinating disease (in MS),
- Motor neuron disease (in middle-aged or elderly).
- Friedreich ataxia (in early age).
- Hereditary spastic paraplegia.
- Subacute combined degeneration of cord
- Syringomyelia
- Anterior spinal artery occlusion
- Tabes dorsalis
- Cervical myelopathy

Causes of spinal cord compression

1. Extradural (lesion in vertebral column):

- Trauma
- Tuberculosis (TB) of spine (Pott disease)
- Lymphoma
- Secondary deposit (elderly)
- Multiple myeloma (elderly)
- Abscess {paravertebral)

2. Intradural (extramedullary and intramedullary):

a. Extramedullary causes (within dura):

- Meningioma
- Neurofibroma
- Secondary deposit (elderly)
- Lymphoma
- Leukaemia

CS CamScanner

b. Intramedullary causes:

- Glioma
- Ependymoma
- Syringomyelia
- Haematomyelia

Causes of spastic paraplegia due to cerebral lesion

- Parasagittal meningioma
- Thrombosis of superior longitudinal sinus
- Thrombosis of unimpaired anterior cerebral artery
- Multiple bilateral cerebral infarctions
- Hydrocephalus
- Trauma
- In children, cerebral palsy (usual lesion is bilateral parasagittal cortical lesion)

Causes of noncompressive spastic paraparesis

- Amyotrophic lateral sclerosis
- Subacute combined degeneration
- Transverse myelitis
- Multiple sclerosis
- Friedreich's ataxia
- Syringomyelia
- Vascular disease of the cord
- Hereditary spastic paraplegia
- Tropical spastic paraplegia
- Postvaccination
- Syphilitic amyotrophy
- Non metastatic manifestation of malignancy
- Radiation myelopathy

Causes of spastic paraparesis with dorsal column signs

- Cervical myelopathy
- Friedreich's ataxia

- Multiple sclerosis
- Taboparesis
- Subacute combined degeneration

Causes of spastic paraparesis according to the type of lesion:

Demyelinating: MS, acute demyelinating encephalomyelitis (ADEM)

Inflammatory: Sarcoidosis, postviral, postvaccinal

Infective: HIV, Herpes zoster, Herpes simplex, syphilis

Degenerative: MND, familial spastic paraplegia (FSP), SCD, Friedreich ataxia (FA)

Vascular: Anterior spinal artery infarction, intramedullary haemorrhage, sp arteriovenous malformation (AVM)

Toxic: Lathyrism, radiation

Causes of absent ankle jerk, but extensor plantar response

- Subacute combined degeneration of the spinal cord.
- Friedreich ataxia
- MS
- Taboparesis
- Diabetes mellitus with cervical myelopathy
- Peripheral neuropathy and stroke
- Motor neuron disease

Causes of flaccid paraplegia

- GBS/CIDP
- Motor neuropathy due to any cause
- Tabes dorsalis
- Friedreich ataxia
- Progressive muscular atrophy
- Acute inflammatory demy el mating polyradiculopathy (A1DP)
- Hysterical conversion reaction (HCR)

Causes of motor neuropathy

- Guillain-Barre syndrome/chronic inflammatory demyelinating polyneuropathy (CIDP)

- Porphyria
- Chronic lead poisoning
- Diabetic amyotrophy
- Diphtheria
- Drugs (Dapsone)
- Charcot-Marie-Tooth disease

Causes of sensory motor polyneuropathy

- Alcoholism
- Diabetes mellitus.
- Nutritional deficiency (B d , B g , B n , folic acid; pantothenic acid and vitamin E)
- Hypothyroidism
- Uremia
- Sarcoidosis
- Vasculitis
- Paraneoplastic
- CIDP/GBS
- Drugs (vinblastin, vincristine; phenytoin, amiodarone, statins, cisplatin and gold).
- Infections (leprosy).
- Idiopathic.
- Hereditary motor and sensory neuropathy

Causes of sensory neuropathy

- Alcohol
- Diabetes mellitus
- Hypothyroidism
- Leprosy
- Sarcoidosis
- Deficiency of vitamins B6 and B 12
- Uremia
- Paraneoplastic

- Vasculitis
- Drugs (isoniazid, metronidazole, hydralazine, chloroquine)
- Amyloidosis
- Hereditary sensory neuropathy
- HIV
- Lyme disease
- Multiple myeloma

Causes of painful neuropathy

- Diabetes mellitus
- Deficiency of vitamins
- Alcoholism
- paraneuplastic
- Porphyria.
- Arsenic or thallium poisoning

Causes of demyelination

- Guillain - Barre syndrome/CIDP
- Multiple myeloma
- Monoclonal gammopathy of undetermined significance (MGUS)
- Hereditary sensory motor neuropathy type 1 and type 3
- Diphtheria
- Diabetes mellitus
- Refsum disease
- HIV
- Multifocal motor neuropathy
- POEMS syndrome

Causes of axonal degeneration:

- Toxic neuropathy (alcohol and drugs)
- Diabetes mellitus
- Paraneoplastic
- IgG paraproteinaemia

- Hereditary
- Vitamin deficiency

Cause of mononeuritis multiplex

- Diabetes
- Polyarteritis nodosa
- Churg strauss syndrome
- Wegner's granulomatosis
- Rheumatoid arthritis
- SLE
- Sjogren's syndrome
- Sarcoidosis
- Lymphoma
- Carcinoma
- Amyloidosis
- Lyme disease
- Leprosy

Causes of thickened peripheral nerves

- Amyloidosis
- Leprosy
- Acromegaly
- Neurofibromatosis
- Hereditary motor and sensory neuropathy (HMSN)

Causes of Lhermitte's sign

- Multiple sclerosis
- Cervical myelopathy
- Cervical cord tumor
- Subacute combined degeneration of cord

Causes of downbeat Nystegmus

- Arnold Chiari malformation

- Tumors
- Syringobulbia
- Spinocerbeller degeneration
- Vertebrobasilar infarction
- Wernicke's encephalopathy

Causes of upbeat Nystegmus

- Lesion at anterior vermis of cerebellum
- Organophosphate poisoning
- Anticonvulsants
- Wernicke's encephalopathy
- Encephalitis
- Meningitis

Causes of upgaze palsy

- Progressive supranuclear palsy
- Graves' ophthalmopathy
- Myasthenia gravis
- Miller-Fisher syndrome
- Muscular dystrophy
- Chronic progressive external ophthalmoplegia
- Parinaud syndrome

Causes of wasting of the small muscles of the hand

- MND
- Syringomyelia
- Charcot-Marie-Tooth disease
- Friedrich's ataxia
- Cervical cord tumors
- Meningovascular syphilis
- Polneuroathy (motor and sensory)
- Distal spinal muscular atrophy

- Old poliomyelitis
- Cervical spondylosis
- Cervical rib
- Pancoast tumour
- Neurofibroma
- Following arthritis
- Myopathy (dystrophia myotonita)
- Distal muscular dystrophies
- Median and ulnar nerve palsy
- Disuse atrophy
- Cachexia

ses of wasting in one hand

- Cervical cord compression or cervical rib
- Brachial plexus lesion (trauma and tumour).
- Pancoast tumour
- Old poliomyelitis
- Cerebral palsy
- Leprosy

ses of pes cavus

lateral:

- Malunion of calcaneal or talar fracture
- burns
- sequalae of compartment syndrome
- Poliomyelitis
- spinal trauma
- spinal cord tumors

lateral:

- Friedreich's ataxia.
- Muscular dystrophies
- Spinal muscular atrophy

- Cerebral palsy
- Syringomyelia
- Hereditary spastic paraparesis
- Charcot-Marie-Tooth disease
- Spinal cord tumors
- Spinocerebellar degeneration

Causes of fasciculation:

- Motor neuron disease
- After exercise in healthy adult
- Electrolyte imbalance (hypokalemia, hypomagnesemia)
- Thyrotoxic myopathy
- Cervical spondylosis
- Syringomyelia
- Hereditary motor and sensory neuropathy
- Acute poliomyelitis
- Meuralgic amyotrophy
- Syphilitic amyotrophy
- After tensilon test
- Spinal muscular atrophy
- Drugs (clofibrates, salbutamol)

Causes of Argyll Robertson pupils

- Neurosyphillis (most common cause)
- Diabetes
- Midbrain lesion
- Lyme disease
- Wernicke's encephalopathy
- Brainstem encephalitis

Causes of relative afferent pupillary defect

- Optic neuritis

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- Central retinal vein occlusion
- Central retinal artery occlusion
- Severe ischemic diabetic retinopathy
- Retinal detachment
- Severe macular degeneration
- Retinal infarction

Causes of mydriasis

- 3rd nerve palsy
- Myotonic pupil
- Mydriatic eye drops
- Perinaud syndrome
- Drugs (atropine, TCA, anti-histamine, cocaine)
- Carbon monoxide poisoning
- Deep coma or death

Causes of miosis

- Infancy or advanced age
- Miotic eye drops
- Argyll Robertson pupil
- Horner's syndrome
- Pontine hemorrhage
- Drugs (opiates, benzodiazepines, clonidine, phenothiazine)
- Organophosphate poisoning
- Iritis

Causes of unilateral ptosis

- III nerve palsy
- Horner's syndrome
- Myasthenia gravis
- Congenital

Causes of bilateral ptosis

- Myasthenia gravis
- Myotonic dystrophy
- Tabes dorsalis
- Bilateral Horner's syndrome
- Chronic progressive external ophthalmoplegia
- Occulopharyngeal muscular dystrophy
- Nuclear III nerve palsy
- Miller Fisher syndrome
- Congenital

Causes of sensorineural deafness

Unilateral:

- Cerebellopontine angle tumor

Bilateral:

- Degenerative changes (presbycusis)
- Amphotericin
- Aminoglycosides
- Loop diuretics
- Mumps
- Rubella

Causes of combined cerebellar, pyramidal and dorsal column signs

- MS
- Friedreich ataxia
- Spinocerebellar degeneration
- Syphilitic meningomyelitis
- Arnold-Chiari malformation

Causes of chorea

Inherited

- Huntington chorea

- Wilson disease
- benign familial chorea
- Paroxysmal choreoathetosis
- neuroacanthocylosis
- spinocerebellar ataxia

quired

- Sydenham chorea
- SLE
- Polycythaemia rubra vera
- Thyrotoxicosis
- Hypoparathyroidism with hypocalcemia
- Senile chorea
- Dopamine antagonists
- Chorea gravidarum
- Oral contraceptive pills
- Encephalitis
- Anti phospholipid syndrome
- Myeloproliferative disorders.
- Kernicterus
- Cerebral birth injury
- Cerebral trauma
- Creutzfeldt-lakob disease

isorder of lower motor neuron lesion

- Motor neuron disease
- Brainstem vascular disease
- Syringobulbia
- Brainstem tumors
- Poliomyelitis
- Guillain-Barre syndrome

- Neurosyphillis
- Subacute meningitis

Disorder of neuromuscular junction

- Botulism
- Myasthenia gravis

Disorder of muscle

- Muscular dystrophies
- Polymyositis

Causes of pseudobulbar palsy

- Motor neuron disease
- Bihemispheric vascular disease
- Brainstem tumors
- Multiple sclerosis
- Trauma

Causes of Bell's (LMN) palsy

Idiopathic (95%)

Structural lesion

- Brainstem: demyelination, stroke, tumors
- Cerebellopontine angle : acoustic neuroma
- Middle ear: infection
- Parotid gland : infection, tumors, surgery
- Mononeuritis multiplex

Causes of bilateral facial palsy

- Guillain-Rarre syndrome (GBS)
- Sarcoidosis
- Bilateral Bell palsy (rare)
- Bilateral parotid disease
- Lyme disease
- Any cause of mononeuritis multiplex (rare)

Causes of bilateral facial weakness

- Bilateral facial palsy
- Myasthaeni a gravis
- Myopathy (fascio-scapulo-humeral myopathy and myotonic dystrophy)

How to locate the site of lesion in LMN facial palsy and what are the causes?

Lesion in pons;

- Associated VIth nerve palsy (lateral rectus muscle of eye) and Vth nerve palsy
- If corticospinal tract is involved, there is contralateral hemiplegia. (Causes—vascular, demyelinating disease, tumour and syringobulbia)

Cerebellopontine angle lesion:

- Usually associated with Vth, VIth and VIIth nerve palsy and also cerebellar signs. (Causes—acoustic neuroma, neurofibroma and meningioma.)

Lesion in petrous part of temporal bone:

- Associated with loss of taste in anterior two-thirds of the tongue and also hyperacusis. (Causes'—Bell palsy, Ramsay Hunt syndrome, trauma and chronic suppurative otitis media (CSOM)].

Lesion in face:

- Parotid tumour, sarcoidosis, and trauma or surgery of parotid gland.

Causes of VIth nerve palsy

- Raised intracranial pressure is the commonest cause, due to stretching of the nerve in its long intracranial course (a false localising sign
- Brainstem lesion (vascular, neoplastic and MS).
- Cavernous sinus lesion (tumour, thrombosis, infection, aneurysm).
- Trauma.
- Any cause of mononeuritis multiplex.
- Idiopathic (common).
- Subacute meningitis (due to fungal, tuberculous, lymphomatous, carcinomatous).

Causes of bilateral VIth nerve palsy

- Trauma.
- Wernicke encephalopathy.
- Mononeuritis multiplex.

- Raised intracranial pressure (VIth nerve palsy is often associated with VIIth lesion also).

Causes of uniocular permanent sudden-onset visual loss

- Retinal artery occlusion
- Anterior ischaemic optic neuropathy
- Retinal vein occlusion
- Traumatic optic neuropathy

Causes of claw hand:

- Combined ulnar and median nerve lesion: Trauma, leprosy.
- Brachial plexus lesion (C8 and Tl): Cervical rib, thoracic inlet syndrome, Klumpke paralysis.
- Neurological disease: MND, Chareot-Marie-Tooth disease, syringomyelia, intramedullary tumour and polio.

Pseudoclaw hand:

- Dupuytren contracture.
- Volkmann ischaemic contracture.
- Postbum contracture.
- Diabetic cheiroarthropathy.

Causes of carpal tunnel syndrome

- Pregnancy (due to fluid retention, usually in the third trimester).
- Obesity.
- RA.
- Acromegaly.
- Myxoedema.
- CRF on long-term dialysis (due to deposition of |32-microglobulin—an amyloid).
- Tuberculous tenosynovitis,
- Primary amyloidosis.
- Tophaceous gout.
- Drug (oral contraceptive pill).

- Osteoarthrosis of carpus (related to old fracture).
- Idiopathic (common in female, middle-aged and obese. May occur in male with unaccustomed hand use, e.g, house painting).

Causes of clubbing and hypertrophic pulmonary osteoarthropathy

Familial

Thoracic

Tumours:

- Benign or malignant Lung cancer
- Mesothelioma
- Pleural fibroma
- Oesophageal cancer
- Oesophageal leiomyoma
- Thymoma
- Atrial myxoma

Interstitial lung disease:

- Idiopathic pulmonary fibrosis (fibrosing alveolitis)
- Asbestosis

Infections:

- Bronchiectasis
- Empyema
- Lung abscess
- Cystic fibrosis
- Bacterial endocarditis

Arteriovenous shunting:

- AV malformations in the lung
- Cyanotic congenital heart disease

Non-thoracic

- Hepatic cirrhosis
- Coeliac disease

- Ulcerative colitis
- Crohn's disease

Causes of differential dubbing

- PDA with reverse shunt (also there is cyanosis in toes, not in finger calle differential cyanosis),
- Infected abdominal aortic aneurysm.
- Coarctation of abdominal aorta.

Causes of unilateral dubbing

- Idiopathic
- Axillary artery aneurysm.
- Bronchial arteriovenous aneurysm.
- Arteriovenous fistula (dialysis fistula)
- Hemiplegia
- Brachial plexus injury
- Trauma
- Aneurysm of ascending aorta, subclavian or innominate artery.

Causes of clubbing in a single finger

- Trauma (the commonest cause),
- Chronic tophaceous gout.
- Sarcoidosis.

Causes of clubbing with cyanosis

- Fibrosing alveolitis.
- Cyanotic heart disease (Fallot tetralogy).
- Cystic fibrosis.
- Bilateral extensive bronchiectasis.

Causes of asterixis

- Respiratory failure/acidosis (CO2 retention)
- Liver failure
- Renal failure

- Electrolyte disturbance
    - Hypoglycaemia
    - Hypokalaemia
    - Hypomagnesaemia
- Wilson's disease - hepatolenticular degeneration
- Drugs
    - Barbiturates
    - Alcohol
    - Sodium valproate
    - Phenytoin
    - Carbamazepine
    - Metoclopramide
    - Gabapentin
    - Ceftazidime
    - Opioids
- CNS
    - Intracerebral haemorrhage
    - Subdural haematoma
    - Subarachnoid haemorrhage
    - Cerebral ischaemia
    - Cerebral lymphoma

uses of tracheal deviation

wards the side of the lung lesion

- Upper lobe lung colapse
- Upper lobe fibrosis
- Pneumonectomy

way from the side of the lung lesion

- Tension pneumothorax
- Massive pleural effusion

Upper mediastinal mass

- Retrosternal goitre
- Lymphoma
- Lung cancer

Causes of bronchial breath sounds

Common

- Lung consolidation (pneumonia)

Uncommon

- Localized pulmonary fibrosis
- At the top of a pleural effusion
- Colapsed lung (where the underlying major bronchus is patent)

Causes of basal crepitation

1. Unilateral basal crepitation:

- Unilateral bronchiectasis.
- Resolution stage of pneumonia.
- Lung abscess,
- Localized fibrosis of lung.

2. Bilateral basal crepitation:

- Bilateral bronchiectasis.

Causes of bronchiectasis

1. Congenital or hereditary:

- Cystic fibrosis.
- Kartagener syndrome (triad of bronchiectasis, dextrocardia, and sinusit frontal sinus agenesis),
- Primary ciliary dyskinesia (including immotile ciliary syndrome).
- Hypogammaglobulinaemia (of IgA and IgC; it causes recurrent infectio bronchiectasis).
- Yellow nail syndrome.
- Young syndrome (obstructive azoospermia and chronic sinopulmonary infe thought to be due to mercury intoxication).
- Sequestrate segment of lung.

2. Acquired:

- In children, pneumonia complicating measles, whooping cough, primary TB and foreign body.
- In adults, bronchial neoplasm, pulmonary TB, recurrent aspiration or suppurative pneumonia.
- Allergic bronchopulmonary aspergillosis (causes proximal bronchiectasis).

Causes of cavitary lesion in lung

- Tuberculosis.
- Cavitating bronchial carcinoma.
- Pulmonary infarction.
- Fungal infection (histoplasmosis).
- Wegener granulomatosis.
- Rheumatoid nodules.
- Consolidation (Staph aureus).

Causes of multiple cavitary lesions in the lung

- Tuberculosis
- Staphylococcal lung abscess (in children)
- Fungal infection
- Klebsiella.
- Amoebiasis.
- Fibrosing alveolitis or ILD
- Pulmonary oedema.

Causes of double apex beat

- Ventricular aneurysm.
- HCM.

Causes of impalpable apex beat

- Thick chest wall (obesity)
- If the apex is behind the rib.
- Emphysema.
- Pericardial effusion
- Dextrocardia (apex is on the right side).

Causes of diffuse apex beat

- Anterior MI (better seen).
- Occasionally, in left ventricular aneurysm

Causes of epigastric pulsation:

- Normally palpable (lean and thin person)
- RVH.
- Aneurysm of abdominal aorta (expansile pulsation).
- Mass overlying aorta (carcinoma of stomach).
- Pulsatile liver (in TR).
- Suprasternal pulse: Usually arterial (due to aneurysm of aorta and atherosclerosis).

Abnormalities of intensity of the first heart sound

Quiet

- Low cardiac output
- Poor left ventricular function
- Long P-R interval (first-degree heart block)
- Rheumatic mitral regurgitation

Loud

- Increased cardiac output
- Large stroke volume
- Mitral stenosis
- Short P-R interval
- Atrial myxoma (rare)

Variable

- Atrial fibrillation
- Extrasystoles
- Complete heart block

Abnormalities of the second heart sound

Quiet

- Low cardiac output

- Calcific aortic stenosis
- Aortic regurgitation

oud

- Systemic hypertension (aortic component)
- Pulmonary hypertension (pulmonary component)

plit

idens in inspiration (enhanced physiological splitting):

- Right bundle branch block
- Pulmonary stenosis
- Pulmonary hypertension
- Ventricular septal defect

ed splitting (unaffected by respiration):

rial septal defect

idens in expirati+on (reversed splitting):

- Aortic stenosis
- Hypertrophic cardiomyopathy
- Left bundle branch block
- Ventricular pacing

uses of third heart sound

ysiological

- Healthy young adults
- Athletes
- Pregnancy
- Fever

thological

- Large, poorly contracting left ventricle
- Mitral regurgitation

uses of MR

- Chronic rheumatic heart disease

- Mitral valve prolapse.
- Papillary muscle dysfunction (due to acute inferior myocardial infarction).
- Left ventricular dilation
- Rupture++ of chordae tendineae (due to infarction, subacute bacterial endocarditis, trauma or spontaneous).
- Infective endocarditis
- Valvotomy or valvuloplasty.
- Connective tissue diseases (RA and SLE).
- Ankylosing spondylitis.
- Cardiomyopathy
- Marfan syndrome
- pseudoxanthoma elasticum
- Ehlers-Danlos syndrome.

Causes of acute MR

- Trauma or surgery (valvotomy).
- SBE (due to perforation of mitral valve leaflet or chordae).
- Acute MI (due to rupture of papillary muscle).
- Acute rheumatic fever (RE) (due to mitral valvulitis).
- Spontaneous rupture of chorda tendineae or myxomatous degeneration of valve

Causes of MS

1. Chronic rheumatic heart disease (the commonest cause)
2. Other causes are (very rare, do not mention unless asked):
    - Congenital.
    - Calcification of valve (usually in elderly).
    - Carcinoid syndrome
    - SLE
    - Mucopolysaccharidosis
    - Fabry's disease
    - Methysergide therapy
    - Whipple disease

se of aortic stenosis according to age

nts, children and adolescents:

- Congenital aortic stenosis.
- Congenital subvalvular aortic stenosis
- Congenital supravalvular aortic stenosis.

ng adults to middle-aged:

- Calcification and fibrosis of congenitally bicuspid aortic stenosis.

le-aged to elderly:

- Senile degenerative aortic stenosis,
- Calcification of bicuspid valve.
- Rheumatic aortic stenosis.

er causes

- Infective endocarditis
- Hyperuricemia
- Alkaptonuria
- Paget's disease of bones

se of AR according to the site or abnormality

ue to involvement of valve:

- Rheumatic fever.
- Infective endocarditis.
- Bicuspid aortic valve,
- Trauma

ortic root dilatation:

- Marfan syndrome
- Dissecting aneurysm affecting ascending aorta
- Syphilitic aortitis
- Hypertension.
- Trauma
- Seronegative arthritides (ankylosing spondylitis, Reiter syndrome, psoriatic arthropathy).

- SLE
- Rheumatoid arthritis.
- Pseudoxamhoma elasticum.
- Osteogenesis imperfecta.
- Rare causes: Appetite suppressants (like fenfluramine, phentermine)

Causes of acute AR

- Acute bacterial endocarditis.
- Acute RF (due to valvulitis).
- Dissecting aneurysm affecting ascending aorta, Trauma,

Clinical signs of severe aortic stenosis

- Low volume pulse
- Slow rising pulse
- Narrow pulse pressure
- Heaving apex
- Systolic thrill
- Reversed splitting of second heart sound
- Soft or absent aortic component of second heart sound
- Fourth heartsounds
- Signs of pulmonary hypertension
- Sign of pulmonary congestion

Clinical signs of severity of mitral stenosis

- Early opening snap (lost with calcified leaflets)
- Increasing length of murmur

Clinical signs of severe mitral regurgitation

- Soft first heart sound
- Third heart sound
- Fourth heart sound
- Displaced apex beat
- Precordial thrill

- Mid-diastolic flow murmur
- Widely split second heart sound
- Signs of pulmonary hypertension
- Signs of heart failure

ical signs of severe aortic regurgitation

- Wide pulse pressure
- Long duration of the diastolic murmur
- Third heart sound
- Austin flat murmur
- Signs of pulmonary hypertension
- Signs of heart failure

uses of proptosis:

- Graves disease
- Cavernous sinus thrombosis
- Cortico-cavernous fistula
- Orbital cellulitis
- Retro-orbital tumor (lymphoma, leukemia, meningioma, metastasis)
- Retro-orbital granuloma (Wegner's, sarcoidosis)
- Trauma

auses of gynecomastia

- Puberty
- Cirrhosis
- Hypogonadism
- Testicular tumors
- Graves disease
- Hemodialysis
- Drugs (anti-androgen, cimetidine, spironolactone, digoxine, ketoconazole, imatinib)

Causes of tall stature:

- Familial
- Klinefelter's syndrome
- Marfan's syndrome
- Homocystinuria
- MEN 2B
- Precocious puberty
- Hyperthyroidism

Causes of webbed neck

- Turner's syndrome
- Noonan's syndrome
- Klippel-Fleil syndrome
- Watson's syndrome

Causes of monoarthritis

- Septic arthritis
- Crystal arthritis (gout/pseudogout)
- Spondyloarhthrtis
- Mycobacterial arthritis
- Fungal arthritis
- Osteoarthritis
- Stills disease
- Rheumatic fever
- Lyme disease

Causes of dural ectasia

- Marfan's syndrome
- Neurofibromatosis type 1
- Ehlers-Danlos syndrome
- Ankylosing spondylitis
- Trauma

- Scoliosis
- Tumors

almar erythema

- Hypercapnia
- Rheumatoid arthritis
- Thyrotoxicoses
- Pregnancy
- Fever
- Exercise

um hyperplasia

- Cyclosporine
- Phenytoin
- acute myeloid leukaemia

luehrckes lines

- Nephrotic syndrome
- Liver disease
- Enteropathy

oilonychia

- Trauma
- Chemotherapy
- radiation therapy
- nutritional deficiency

auses of High-arched palate

- Friedreich ataxia.
- Marfan syndrome.
- Homocystinuria.
  - Turner syndrome.
  - Tuberous sclerosis.

auses of hypoalbuminaemia and leukonychia

educed albumin synthesis

- Chronic liver disease

Urinary protein loss

- Nephrotic syndrome

Protein-losing enteropathy

- Crohn's disease
- Ulcerative colitis
- Ménétrier's disease of the stomach
- Coeliac disease
- Intestinal lymphoma
- Bacterial overgrowth
- Radiation damage

Causes of Purpura

Platelet defect

- Thrombocytopenia
- Uremia
- Chronic liver disease
- Insecticides

Capillary defect

- Age
- Steroids
- Vasculitis
- Cholesterol emboli
- Ehlers-Danlos syndrome
- Pseudoxanthoma elasticum
- Hereditary hemorrhagic telangiectasia
- Scurvy
- Amyloidosis
- Venous stasis

Coagulation defect

- Antithrombotic therapy

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- Von-Will brand's disease
- Haemophillia
- DIC

ses of lichen planus

- Idiopathic
- Drugs (B-blockers, ACE-I, NSAIDS, Gold)
- Hepatitis C

rmalities of urine colour

nge-brown

- Conjugated bilirubin
- Rhubarb, senna
- Concentrated normal urine, e.g. very low fluid intake

-brown

- Blood, myoglobin, free haemoglobin, porphyrins
- Beetroot, blackberries
- Drugs: rifampicin, metronidazole, warfarin

wn-black

- Conjugated bilirubin
- Drugs: L-dopaHomogentisic acid (in alkaptonuria or ochronosis)

e-green

- Drugs/dyes, e.g. propofol, fluorescein

ses of periorbital oedema

- Allergic eye disease
- Thyroid eye disease
- Orbital cellulitis
- Nephrotic syndrome
- Heart failure
- Angio-oedema

Nail fold infarction

- SLE
- Dermatomyositis.
- Systemic sclerosis.
- Rheumatoid arthritis (RA).
- Polyarteritis nodosa.

Splinter haemorrhage

- Trauma (the commonest).
- SLE.
- Septicaemia.
- Collagen disease (vasculitis): SLE, RA and polyarteritis nodosa,
- Others: Haematological malignancy, severe anaemia, psoriasis.

Half-and-half nail:

- CRF (the commonest cause).
- Cirrhosis of liver.
- Occasionally, in normal person.
- Red half moon occurs in congestive cardiac failure (CCF).

Nail fold telangiectasia

- SLE.
- Systemic sclerosis.
- Dermatomyositis.
- Mixed connective tissue disease (MCTD),
- Raynaud phenomenon.

Beau line

- Chronic illness (chronic infection, malignancy and collagen disease).
- Prolonged fever.
- Pneumonia
- Coronary artery disease
- Others: Cachexia, malnutrition, psychiatric illness, use of cytotoxic drugs.

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nycholysis

- Psoriasis (the commonest).
- Fungal infection.
- Thyrotoxi cos is (Plummer sign}.
- Idiopathic
- Occasionally drugs (tetracycline and psoralen)
- Porphyria
- Trauma or faulty manicure

ee's line

- Chronic arsenic poisoning.
- CRF
- After chemotherapy and severe illness

ail pitting

- Psoriasis
- Alopecia areata
- Atopic eczema
- Pityriasis rosacea

Terry nail

- Old age (normally present in elderly).
- Cirrhosis of liver
- CCF
- Hyperthyroidism
- Malnutrition
- Renal failure

Generalized lymphadenopathy

- Haematological malignancy: Lymphoma, acute lymphoblastic leukaemia (ALL. chronic lymphocytic leukaemia (CLL)
- Viral fever: Infectious mononucleosis, cytomegalovirus (CMV) infection, human immuno deficiency virus (HIV).

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- Other infections: Disseminated tuberculosis, brucellosis, and toxoplasmosis.
- Sarcoidosis
- SLE
- Drugs: Phenytoin or diphenylhydantoin

Generalized lymphadenopathy with arthritis

- Juvenile idiopathic arthritis (J1A) (early age).
- SLE.
- Sarcoidosis
- Viral-fever-like infectious mononucleosis, CMV
- infection*
- Brucellosis.

Erythema nodosum

Bacterial infections

- Streptococcal beta-haemolyticus infection
- Primary pulmonary TB
- Leprosy
- Mycoplasma
- Chlamydia
- Syphilis
- Salmonella
- Yersinia
- Campylobacter

Viral infections

- CMV
- EBV

Fungal infections

- Coccidiomycoisis
- Histoplasmosis
- Blastomycosis

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Drugs

- Sulphonamide
- Penicillin
- Tetracyclins
- Gold
- oestrogen containing oral contraceptive pill
- salicylates
- barbiturates
- sulphonylureas
- bromides and iodides

Others:

- Sarcoidosis
- Inflammatory bowel disease (Crohn disease, ulcerative colitis).
- Behcet syndrome
- SLE
- Lymphoma

Causes of livedo reticularis

- Physiological
- Idiopathic
- Primary livedo reticularis
- Antiphospholipid syndrome
- Cryoglobulinaemia
- Polycythemia rubra vera
- Essential thrombocytosis
- Waldenstrom's macroglobulinemia
- Cold agglutinins
- SLE
- Polyarteritis nodosa
- Wegner's granuloma

- Churg-strauss syndrome
- Cholesterol embolization
- Septic embolization

Causes of Dupuytren contracture

- Cirrhosis of liver
- Alcoholism
- Prolonged use of antiepileptic drug (phenytoin).
- Manual worker (gardener) and chronic vibration injury.
- Traumatic
- Familial
- Diabetes mellitus (diabetic cheiroarthropathy, confuses with systemic sclerosis).
- May be associated with tuberculosis.
- Retroperitoneal fibrosis.
- Peyronie disease.
- Idiopathic (in many cases).

Skin lesions in diabetes

- Infections (boils, carbuncles, cellulitis)
- Ulceration and gangrene
- Lipohypertrophy
- Lipoatrophy
- Eruptive xanthomatas
- Xanthelasmas
- Acanthosis nigricans
- Erythema nodosum
- Erythema multiforme
- Vitiligo
- Alopecia
- Granuloma annulare
- Diabetic dermopathy
- Diabetic rubeosis
- Necrobiosis lipoidica

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Chapter – 8

Overview of Clinically Important Topics

INFECTIVE ENDOCARDITIS

odified Duke Criteria for diagnosis of infective endocarditis

efinite IE

thologic criteria

- Microorganism: demonstrated by culture or histology in a vegetation, or in a vegetation that has embolized, or in an intracardiac abscess OR
- Pathologic lesions: vegetation or intracardiac abscess, confirmed by histology showing active endocarditis

linical criteria (given blow)

- 2 major criteria OR
- 1 major and 3 minor criteria OR
- 5 minor criteria

ossible IE

- 1 major criterion and 1 minor criterion OR 3 minor criteria

ejected IE

- Firm alternate diagnosis for manifestations of endocarditis OR
- Resolution of manifestations of endocarditis, with antibiotic therapy for four days or less OR
- No pathologic evidence of infective endocarditis at surgery or autopsy after antibiotic therapy for four days or less
- Does not meet criteria for possible infective endocarditis, as above

ajor criteria

ositive blood cultures for IE

ypical microorganism for infective endocarditis from two separate blood cultures

- Viridans streptococci

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- Streptococcus gallolyticus (formerly S. bovis), including nutritional variant strains (Granulicatellaspp and Abiotrophiadefectiva)
- HACEK group - Haemophilusspp, Aggregatibacter Actinobacillusactinomycetecomitants), Cardiobacteriumhom and Kingellakingae.
- Staphylococcus aureus
- Community-acquired enterococci, in the absence of a primary focus; OR

Persistently positive blood culture, defined as recovery of a microorganism consistent with IE from:

- Blood cultures drawn more than 12 hours apart OR
- All of three or a majority of four or more separate blood cultures with first and last drawn at least one hour apart

Single positive blood culture for Coxiellaburnetii or antiphase I IgG antibody titer >1:800*

Evidence of endocardial involvement

Positive echocardiogram for IE

- TEE recommended in patients with prosthetic valves, rated at least "possible IE" by clinical criteria, or complicated IE [paravalvular abscess]; TTE as first test in other patients*
- Definition of positive echocardiogram
    - Oscillating intracardiac mass, on valve or supporting structures, or in the path of regurgitant jets, or on implanted material, in the absence of an alternative anatomic explanation OR Abscess OR
    - New partial dehiscence of prosthetic valve

New valvular regurgitation

- Increase in or change in preexisting murmur not sufficient

Minor criteria

- Predisposition - predisposing heart condition or intravenous drug use
- Fever - 38.0°C (100.4°F)
- Vascular phenomena - major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, Janeway lesions
- Immunologic phenomena - glomerulonephritis, Osler's nodes, Roth spots, rheumatoid factor

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- Microbiologic evidence - positive blood culture but not meeting major criterion as noted previously (excluding single positive cultures for coagulase-negative straphylococci and organisms that do not cause endocarditis) OR serologic evidence of active infection with organism consistent with IE

ggested regimens for therapy of infective endocarditis according to British rdiac Society

spected organism Organism	Antibiotics
tive valve endocarditis due penicillin-susceptible idans streptococci and eptococcus bovis	Benzylpenicillin 1.2 g every 4 h IV for 4 weeks or Ceftriaxone 2 g per 24 h IV/IM for 4 weeks
	Either benzylpenicillin 1.2 g every 4 h IV for 2 weeks or Cefriaxone 2 g per 24 h IV/IM for 2 weeks **plus** Gentamicin § 1 mg/kg every 12 h IV for 2 weeks
tive valve endocarditis due strains of viridans eptococci and eptococcus bovis relatively istant to penicillin	Benzylpenicillin 2.4 g every 4 h IV for 4 to 6 weeks **plus** Gentamicin 1 mg/kg every 12 h IV for 2 weeks
tive valve endocarditis due staphylococcus - thicillin sensitive	Flucloxacillin 2 g IV every 4 to 6 hours for 4 weeks
tive valve endocarditis due staphylococcus - thicillin resistant	Vancomycin 1 g IV every 12 h for 4 weeks **plus** Rifampin 300 to 600 mg PO every 12 h for 4 weeks
tive valve endocarditis due HACEK microorganism	Either AmpicillinΔ 2 g IV every 4 to 6 h for 4 wksor Ceftriaxone• 2 to 4 g per 24 h IV or IM in 1 dose for 4 wks **plus** Gentamicin◊ 1 mg/kg IV every 8 to 12 h for 2 wks
lture-negative native valve docarditis	Vancomycin 15mg/kg IV every 12 h for 6 wks plus Gentamicin 3-5 mg/kg IV or IM per 24 h in 2 or 3 qually divided doses for 2 wks

Prosthetic valve endocarditis due to staphylococcus-Methicillin-susceptible strains	Oxacillin or cloxacillin or flucloxacillin 12g per 24 h IV in 4 to 6 equally divided doses for ≥6 weeks or Vancomycin§ 30 mg/kg per 24 h IV in 2 equally divided doses for ≥6 weeks **plus** Rifampin 1200 mg per 24 h IV or PO in 2 equally divided doses for ≥6 weeks **plus** Gentamicin 3 mg/kg per 24 h IV or IM in 2 or 3 equally divided doses for the first 2 weeks
Prosthetic valve endocarditis due to staphylococcus-Methicillin resistant strains	Vancomycin 30 mg/kg per 24 h IV in 2 equally divided doses for ≥6 weeks **plus** Rifampin 1200 mg per 24 h IV or orally in divided doses for ≥6 weeks **plus** Gentamicin 3 mg/kg per 24 h IV or IM in 3 equally divided doses for first 2 weeks
Prosthetic valve endocarditis due to penicillin-susceptible viridans streptococci and Streptococcus bovis	Either aqueous penicillin G 12 to 18 million units per 24 h IV in 6 equally divided doses for 6 wks or Ceftriaxone 2 g per 24 h IV or IM in 1 dose for 6 wks or Vancomycin30 mg/kg per 24 h IV in 2 equally divided doses for 6 wks`
Prosthetic valve endocarditis due to strains of viridans streptococci and Streptococcus bovis relatively or fully resistant to penicillin G	Vancomycin 30 mg/kg per 24 h IV in 2 doses for 4 wks plus Gentamicin 3 mg/kg per 24 h IV or IM in 1 dose for first 2 wks (see text)
Prosthetic valve endocarditis due to HACEK microorganism	Combination therapy with: ampicillin 12 g per 24 h IV in 4 equally divided doses for 6 wks PLUS gentamicin 3 mg/kg per 24 h IV or IM divided in 2 or 3 doses for 6 wks

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ACUTE RHEUMATIC FEVER

gnostic criteria for Acute rheumatic fever (reviewed in 2002)

gnosis n the setting of group A streptococcal infection require:

- Two major manifestations or
- one major and two minor manifestations

e five major manifestations are:

- Migratory arthritis (predominantly involving the large joints)
- Carditis and valvulitis (eg, pancarditis)
- Central nervous system involvement (eg, Sydenham chorea)
- Erythema marginatum
- Subcutaneous nodules

e four minor manifestations are:

- Arthralgia
- Fever
- Elevated acute phase reactants [erythrocyte sedimentation rate (ESR), C-reactive protein (CRP)]
- Prolonged PR interval

esumptive diagnosis of acute rheumatic fever can be made without strict herence to the above criteria:

- Chorea as the only manifestation.
- Indolent carditis as the only manifestation in patients who come to medical attention months after acute group A streptococcal infection.
- Recurrent rheumatic fever in patients with a history of rheumatic fever or rheumatic heart disease.

iagnostic criteria for Peripartum cardiomyopathy

e diagnosis of PPCM has been based upon four clinical criteria:

- Development of cardiac failure in the last month of pregnancy or within five months of delivery.
- Absence of an identifiable cause for the cardiac failure
- Absence of recognizable heart disease prior to the last month of pregnancy.

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- LV systolic dysfunction (e.g., LVEF <45 percent or a reduced shortening fraction).

RHEUMATOID ARTHRITIS

ACR criteria for Rheumatoid arthritis:

A score of ≥ 6 out of 10 is needed for classification of a patient as having definite Rheumatoid arthritis.

A. Joint involvement

One large joint	0
Two to 10 large joints	1
One to three small joints (with or without involvement of large joints)	2
Four to 10 small joints (with or without involvement of large joints)	3
> 10 joints (at least one small joint)	5

B. Serology (at least one test result is needed for classification)

Negative RF *and* negative ACPA	0
Low positive RF *or* low positive ACPA	2
High positive RF *or* high positive ACPA	3

C. Acute phase reactants (at least one test result is needed for classification)

Normal CRP and normal ESR	0
Abnormal CRP or normal ESR	1

D. Duration of symptoms

< six weeks	0
≥ six weeks	1

SYSTEMIC LUPUS ERYTHROMATOSIS

SLICC criteria for diagnosis of SLE

Requirement :≥ 4 criteria (at least one clinical and one laboratory criteria) or biopsy proven lupus nephritis with positive ANA or anti-dsANA.

Clinical criteria:

1. Acute cutaneous lupus
   - Malar rash

- o Subacute cutaneous Lupus erythematosus (SCLE) rash
- o Palpable purpura or urticarial vasculitis
- o Photosensitivity

2. Chronic cutaneous lupus
   - Discoid lupus erythematosus (DLE) rash or hypertrophic Lupus rash
3. Oral or nasal ulcers
4. Non-scarring alopecia
5. Arthritis
6. Serositis
   - Pleurisy and/or pericarditis
7. Renal involvement
   - proteinuria≥ 3+ or ≥ 500 mg/day or urinary casts
   - Biopsy-proven nephritis compatible with SLE
8. Neurologic involvement
   - Psychosis and/or seizure and/or acute confusion
9. Hemolytic anemia
10. Leukopenia
    - WBC count < 4000/mm3 or lymphocyte count < 1500/mm3 on ≥ 2 occasions or WBC count < 4000/mm3 along with lymphocyte count < 1500/mm3 in one occasion
11. Thrombocytopenia
    - <100,000

munological criteria:

1. ANA
2. Anti-dsDNA
3. Anti-sm
4. Anti-phospholipid ab
5. Low complement level (C3,C4, CH50)

TNM staging system for lung cancer

Primary tumor (T)

T1: Tumor ≤3 cm diameter, surrounded by lung or visceral pleura, without invasion more proximal than lobar bronchus

- T1a: Tumor ≤2 cm in diameter
- T1b: Tumor >2 cm but ≤3 cm in diameter

T2: Tumor >3 cm but ≤7 cm, or tumor with any of the following features:

  - Involves main bronchus, ≥2 cm distal to carina
  - Invades visceral pleura
  - Associated with atelectasis or obstructive pneumonitis that extends to the hilar region but does not involve the entire lung

- T2a Tumor >3 cm but ≤5 cm
- T2b Tumor >5 cm but ≤7 cm

T3: Tumor >7 cm or any of the following:

  - Directly invades any of the following: chest wall, diaphragm, phrenic nerve, mediastinal pleura, parietal pericardium, main bronchus <2 cm from carina (without involvement of carina)
  - Atelectasis or obstructive pneumonitis of the entire lung
  - Separate tumor nodules in the same lobe

T4: Tumor of any size that invades the mediastinum, heart, great vessels, trachea, recurrent laryngeal nerve, esophagus, vertebral body, carina, or with separate tumor nodules in a different ipsilateral lobe

Regional lymph nodes (N)

N0: No regional lymph node metastases

N1: Metastasis in ipsilateralperibronchial and/or ipsilateralhilar lymph nodes and intrapulmonary nodes, including involvement by direct extension

N2: Metastasis in ipsilateralmediastinal and/or subcarinal lymph node(s)

N3: Metastasis in contralateral mediastinal, contralateral hilar, ipsilateral or contralateral scalene, or supraclavicular lymph node(s)

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istant metastasis (M)

0: No distant metastasis

- M1 Distant metastasis
  - M1a Separate tumor nodule(s) in a contralateral lobe; tumor with pleural nodules or malignant pleural or pericardial effusion
  - M1b Distant metastasis (in extrathoracic organs)

tage groupings

tage I:

- Stage IA: T1a-T1b N0 M0
- Stage IB: T2a N0 M0

tage II:

- Stage IIA:
  - T1a/T1b/T2a N1 M0
  - T2b N0 M0
- Stage IIB
  - T2b N1 M0
  - T3 N0 M0

tage III:

- Stage IIIA
  - T1a,T1b,T2a,T2b N2 M0
  - T3 N1,N2 M0
  - T4 N0,N1 M0
- Stage IIIB
  - T4 N2 M0
  - Any T N3 M0

Stage IV:

- Any T Any N M1a or M1b

Staging for small cell lung carcinoma:

- **Limited disease** is defined as disease confined to the ipsilateralhemithorax and within a single radiotherapy port (corresponding in part to TNM stages I through IIIB.

- **Extensive disease** is defined as evident metastatic disease outside the ipsilateralhemithorax.

Treatment protocols for small-cell carcinoma of the lung

Carboplatin plus etoposide

- Cisplatin (60 mg/m2) plus etoposide
- Cisplatin (80 mg/m2) plus etoposide

Treatment protocols for non-small cell lung cancer

- Carboplatin plus docetaxel
- Carboplatin plus pemetrexed
- Cetuximab, vinorelbine, and cisplatin
- Cisplatin plus pemetrexed
- Gemcitabine plus carboplatin
- Gemcitabine plus cisplatin (GC)
- Paclitaxel carboplatin
- Paclitaxel, carboplatin, andbevacizumab
- Vinorelbine plus cisplatin

Management of stage I and stage II non-small cell lung cancer

SURGERY (definite treatment for clinical stage I and II)

- Lobectomy
- Limited (sublobar) resection
- Videoassistedthoracoscopic surgery
- Mediastinal lymph node dissection

NONSURGICAL APPROACHES (if not suitable or refused surgery)

- Definitive RT
- Stereotactic body radiation therapy
    - Heavy particle irradiation
- Radiofrequency ablation
- Cryoablation

ADJUVANT THERAPY

- Adjuvant chemotherapy (role not well known)

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- Postoperative RT- prevent local recurrence following resection of a NSCLC

lanagement of stage III non-small cell lung cancer

- Surgeryif resectable stage IIIA followed by concurrent chemoradiotherapy.
- Concurrent chemoradiotherapy forunresectable stage III disease

ontraindication of surgery in lung carcinoma:

- Malignant pleural effusion
- Superior vena cava obstruction

HEPATOCELLULAR CARCINOMA

ASL guidelines for diagnosis of hepatocellular carcinoma

or nodules seen on ultrasound that are <1 cm, the EASL guidelines recommend:

- Repeat ultrasound at four months. If the nodule grows, additional investigation is required and dictated by the size of the nodule. If the nodule is stable, ultrasound is repeated at four-month intervals for one year, after which time it can be performed every six months.

or nodules between 1 and 2 cm:

- Obtain a 4-phase CT scan **and/or** dynamic contrast enhanced MRI.
- If there are radiologic hallmarks of HCC (arterial hypervascularity and venous/late phase wash out), then a diagnosis of HCC is made.
- If the radiologic hallmarks of HCC are not seen, a biopsy should be obtained and assessed by an expert pathologist. If the biopsy results are inconclusive a second biopsy is recommended. If the results are still inconclusive, ultrasound should be repeated at four-month intervals to monitor for growth, with a repeat biopsy if there is growth or changes in the nodule's enhancement pattern.

or nodules >2 cm:

- Obtain a 4-phase CT scan **or** dynamic contrast enhanced MRI.
- If there are radiologic hallmarks of HCC (arterial hypervascularity and venous/late phase wash out), then a diagnosis of HCC is made.
- If the radiologic hallmarks of HCC are not seen then a biopsy should be obtained to confirm the diagnosis. If the biopsy results are inconclusive, ultrasound should be repeated at four-month intervals to monitor for growth.

reatment approaches for hepatocellular carcinoma

everal treatment modalities are available, including:

- Liver transplantation

- Radiofrequency ablation (RFA) and microwave ablation
- Percutaneous ethanol or acetic acid ablation
- Transarterial chemoembolization (TACE)
- Radioembolization
- Cryoablation
- Radiation therapy and stereotactic radiotherapy
- Systemic chemotherapy (sorafinib) and molecularly targeted therapies

Barcelona clinic liver cancer staging classification and treatment algorithm

Stage 0: (PST 0, Child-Pugh A)

- Very early stage
    - HCC < 2cm
    - Carcinoma in Situ
        - 1 lesion –

          If normal portal pressure/ serum bilirubin – resection of lesion

          If increased portal pressure/ serum bilirubin- look for any associated comorbidity
            - If no – liver transplant
            - If yes- ablation
        - 3 nodules less than 3cm

          Look for any associated comorbidity
            - If no – liver transplant
            - If yes- ablation

Stage A-C:

Early stage (A):

1 HCC or 3 lesion less than 3cm

- 1 lesion –

  If normal portal pressure/ serum bilirubin – resection of lesion

  If increased portal pressure/ serum bilirubin- look for any associated comorbidity

  - If no – liver transplant
  - If yes- ablation
- 3 nodules less than 3cm

  Look for any associated comorbidity

  - If no – liver transplant
  - If yes- ablation

Intermediate stage (B)

Multi-nodular/ PST 0

- Chemoembolization

Advanced stage (C):

Portal invasion/N1,M1/PST 1-2)

- Sorafinib

Stage D:

Terminal stage (D)

- Best supportive care

LYMPHOMA

nn Arbor staging classification for Hodgkin and non-Hodgkin lymphomas

tage I:

Involvement of a single lymph node region (I) or of a single extralymphatic organ or site (IE)*

tage II

Involvement of two or more lymph node regions or lymphatic structures on the same side of the diaphragm alone (II) or with involvement of limited, contiguous extralymphatic organ or tissue (IIE)

tage III

Involvement of lymph node regions on both sides of the diaphragm (III), which may include the spleen (IIIS) or limited, contiguous extralymphatic organ or site (IIIE) or both (IIIES)

tage IV

Diffuse or disseminated foci of involvement of one or more extralymphatic organs or tissues, with or without associated lymphatic involvement

All cases are subclassified to indicate the absence (A) or presence (B) of the systemic ("B") symptoms of significant unexplained fever (>38 degrees C), night sweats, or unexplained weight loss exceeding 10 percent of body weight during the six months prior to diagnosis.

Clinical stage refers to the extent of disease determined by diagnostic tests following a single diagnostic biopsy. If a second biopsy of any kind is obtained, even if negative, the term pathologic stage is used.

*The designation "E" generally refers to extranodal contiguous extension (ie, proximal or contiguous extranodal disease) that can be encompassed within an irradiation field appropriate for nodal disease of the same anatomic extent. A single extralymphatic site as the only site of disease should be classified as IE, rather than stage IV.

Treatment regimens

Drugs	Malignancy
Rituximab plus cyclophosphamide, doxorubicin, vincristine, and prednisone with 21 days between cycles (R-CHOP-21)	non-Hodgkin lymphoma
Dose-adjusted etoposide, prednisone, vincristine, cyclophosphamide, doxorubicin, and rituximab (daEPOCH-R)	non-Hodgkin lymphoma
Rituximab, ifosfamide, carboplatin, and etoposide (R-ICE)	diffuse large B-cell lymphoma
Fludarabine, cyclophosphamide, and rituximab (FCR)	chronic lymphocytic leukemia
Doxorubicin plus bleomycin, vinblastine, and dacarbazine (ABVD)	Hodgkin lymphoma (stage I-II)
Stanford V (doxorubicin, vinblastine, mechlorethamine, vincristine, bleomycin, etoposide , and prednisone)	Hodgkin lymphoma (stage III, IV)
Escalated or standard BEACOPP (bleomycin , etoposide , doxorubicin , cyclophosphamide , vincristine , procarbazine , and prednisone)	Hodgkin lymphoma(stage III, IV)

- **Pretreatment considerations:**
  - Emesis risk: MODERATE (30 to 90 percent frequency of emesis).
  - Prophylaxis for infusion reactions: Premedicate with acetaminophen and diphenhydramine, with or without an H2 blocker, 30 minutes prior to at least the first and second infusions of rituximab.

- Infection prophylaxis: Primary prophylaxis with hematopoietic growth factors should be considered on an individual basis, particularly for high-risk patients such as those with preexisting neutropenia, advanced disease, poor performance status, or patients over age 65 years.
- Dose adjustment for baseline liver or renal dysfunction: dose should be adjusted
- Hepatitis screening: Patients should be screened for hepatitis B and C prior to starting rituximab, and if positive, considered for hepatitis B prophylaxis.
- Vesicant/irritant properties: Doxorubicin and vincristine are vesicants
- Cardiac screening: LVEF should be evaluated prior to initiation of therapy. Dose alterations should be considered for LVEF <50 percent, and doxorubicin therapy is contraindicated in patients with LVEF <30 percent at initiation. Infusion times and schedule may be adjusted to decrease the risk of cardiotoxicity in individuals at high risk for its development.
- Prophylaxis for tumor lysis syndrome: Due to the significant risk of tumor lysis syndrome during the first cycle in CLL, all patients should be well hydrated prior to and during therapy and receive allopurinol 300 mg orally once daily beginning the day prior to the first dose of rituximab and continuing for 14 days

itoring parameters:

- CBC with differential and platelet count weekly during treatment.
- Assess basic metabolic panel (creatinine and electrolytes) daily during treatment and prior to each subsequent treatment cycle.
- Assay for liver function prior to each treatment cycle.
- Dipstick urine for hematuria daily, on days 4 and 5.
- Carriers of hepatitis B or C should be monitored for clinical and laboratory signs of active infection during and following completion of therapy. Rituximab should be discontinued if reactivation occurs.
- LVEF should be evaluated periodically based on LVEF at initiation of therapy and cumulative dose of doxorubicin.
- Assess serum creatinine, potassium, phosphate, calcium, uric acid, and LDH daily during treatment for patients at risk for tumor lysis syndrome.

gested dose modifications for toxicity:

- Myelotoxicity: Delay treatment cycle until the absolute neutrophil count is greater than 1000/mm 3 and platelet count is greater than 50,000/mm 3. Dose reduction not indicated for intracycle hematologic toxicity.

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- If there is a change in body weight of at least 10 percent, dose should b recalculated for all drugs.
- Neuropathy: Dose adjustment of vincristine may be necessary if the severit of neuropathy persists or worsens. No specific guidelines are available fo dose adjustments.
- Pulmonary toxicity: Bleomycin should be discontinued in patients wit documented or strongly sustpectedbleomycin-induced lung injury

Radiation fields

- Extended-field radiation — Radiation treatment volume includes not only the clinically involved nodes, but also the adjacent, clinically uninvolved sites (eg, mantle field or inverted-Y field).
- Involved-field radiation — Radiation treatment volume encompasses all of the clinically involved regions (eg, mediastinal plus low bilateral supraclavicular field which covers the entire mediastinum).
- Involved-node radiation — Radiation treatment volume includes pre- and post-chemotherapy nodal volumes plus a margin of healthy tissue ≤5 cm

ACUTE MYELOID LEUKEMIA

Classification acute myeloid leukemia - French-American-British (FAB)

Eight categories of AML are recognised:

1. M0 (undifferentiated AML)
2. M1 (myeloblastic - immature)
3. M2 (myeloblastic - mature)
4. M3 (promyelocytic; acute promyelocyticleukaemia; APL)
5. M4 (myelomonocytic)
6. M5 monocytic (gingival hyperplasia)
7. M6 (erythroid)
8. M7 (megakaryoblastic).

Induction therapy for acute myeloid leukemia

Standard dose cytarabine plus an anthracycline (7+3):

It combine a seven-day continuous intravenous infusion of cytarabine (100 or 200 mg/m 2 per day) with a short infusion or bolus of an anthracycline given on days one through three

Post remission therapy:

Two options

1. **Consolidation chemotherapy**
   - High dose cytarabine (HiDAC) for younger adults
   - two cycles of daunorubicin (30 to 45 mg/m 2 for two days) and cytarabine (ara-C, 100 to 200 mg/m 2 per day for five days) for older adults
2. **Allogeneic hematopoietic cell transplantation (HCT)**

CHRONIC MYELOID LEUKEMIA

Treatment of chronic myeloid leukemia in chronic phase

The two main treatment options for patients with newly diagnosed CML in chronic phase are:

- BCR-ABL tyrosine kinase inhibitors (TKIs), such as imatinib ,dasatinib , and nilotinib .
- Allogeneic hematopoietic cell transplantation (HCT).

Aims of therapy and duration of treatment:

The aim of initial therapy with a tyrosine kinase inhibitor (TKI) is to achieve the following milestones:

- Complete hematologic response by three to six months
- Any cytogenetic response by six months
- Major cytogenetic response by 12 months
- Complete cytogenetic response by 18 months

Definitions of hematologic, cytogenetic, and molecular response in chronic myeloid leukemia:

Complete Hematologic response (CHR):

- WBC <10 x 10 9 /L
- Basophils <5 percent
- No myelocytes, promyelocytes, myeloblasts in the differential
- Platelet count <450 x 10 9 /L
- Spleen nonpalpable

Cytogenetic response:

- Complete (CCgR): No Ph+ metaphases
- Partial (PCgR): 1 percent to 35 percent Ph+ metaphases
- Major (MCgR): CCgR plus PCgR
- Minor (mCgR): 36 percent to 65 percent Ph+ metaphases
- Minimal (minCgR): 66 percent to 95 percent Ph+ metaphases
- None (noCgR): >95 percent Ph+ metaphases

Molecular response:

- Complete (CMolR): Undetectable BCR-ABL mRNA transcripts by real time quantitative and/or nested PCR in two consecutive blood samples of adequate quality (sensitivity >10 4)
- Major (MMolR): Ratio of BCR-ABL to ABL (or other housekeeping genes) ≤0.1 percent on the international scale

If the TKI is not tolerated or these milestones have not been achieved or have been lost, therapy should be altered. If these milestones have been realized, therapy with the TKI is continued indefinitely at the same dose as tolerated.

Indications for treatment in CLL:

1) Progressive marrow failure: development or worsening of anemia and/or thrombocytopenia
2) Autoimmune cytopaenias e.g. ITP, AIHA
3) Massive (>10 cm) or progressive lymphadenopathy
4) Massive (>6 cm) or progressive splenomegaly
5) Progressive lymphocytosis: > 50% increase over 2 months or lymphocyte doubling time < 6months
6) Systemic symptoms: weight loss > 10% in previous 6 months, fever >38 C for > 2 weeks, extreme fatigue, night sweats

APLASTIC ANEMIA

Disease severity:

Moderate AA:

- Marrow cellularity <30 percent
- Absence of severe pancytopenia
- Depression of at least two of three blood elements below normal.

evere AA (SAA):

- A marrow biopsy showing <25 percent of normal cellularity, or
- A marrow showing <50 percent normal cellularity in which <30 percent of the cells are hematopoietic and at least two of the following are present: absolute reticulocyte count <40,000/microL; absolute neutrophil count (ANC) <500/microL; or platelet count <20,000/microL.

ery severe AA (vSAA): —

- The patient is considered to have very severe aplastic anemia (vSAA) if the criteria for SAA are met and the ANC is <200/microL

REATMENT OVERVIEW

nder age 20 allogeneic HCT is the treatment of choice

ges 20 to 45

Allogeneic HCT could be the first choice

Immunosuppressivetherapy (ATG and cyclosporine) is second line

ver age 45

Immunosuppressive therapy (ATG and cyclosporine) is recommended.

INFLAMMATORY BOWEL DISEASE

LCERATIVE COLITIS

efinitions of disease extent:

- Ulcerative proctitis refers to disease limited to the rectum
- Ulcerative proctosigmoiditis refers to disease limited to the rectum and sigmoid colon and not involving the descending colon
- Left-sided or distal ulcerative colitis refers to disease that extends beyond the rectum and as far proximally as the splenic flexure
- Extensive colitis refers to disease extending proximal to the splenic flexure but sparing the cecum
- Pancolitis refers to disease extending proximal to the splenic flexure and involving the cecum

Assessment of clinical severity:

Mild – Patients with mild clinical disease have four or fewer stools per day with or without blood, no signs of systemic toxicity, and a normal erythrocyte sedimentation rate (ESR). Mild crampy pain, tenesmus, and periods of constipation are also common, but severe abdominal pain, profuse bleeding, fever, and weight loss are not part of the spectrum of mild disease.

Moderate – Patients with moderate clinical disease have frequent loose, bloody stools (>4 per day), mild anemia not requiring blood transfusions, and abdominal pain that is not severe. Patients have minimal signs of systemic toxicity, including a low grade fever. Adequate nutrition is usually maintained and weight loss is not associated with moderate clinical disease.

Severe – Patients with a severe clinical presentation typically have frequent loose bloody stools (≥6 per day) with severe cramps and evidence of systemic toxicity as demonstrated by a fever (temperature ≥37.5°C), tachycardia (HR ≥90 beats/minute), anemia (hemoglobin <10.5 g/dL), or an elevated ESR (≥30 mm/hour). Patients may have rapid weight loss.

Medical therapy of active ulcerative colitis:

Role	Medication		Daily dose
Induction of remission	Topical (rectal) mesalamine	Suppository	1 gram (one suppository) twice daily
		Retention enema	4 grams (one 60 mL unit) twice daily
	Topical (rectal) glucocorticoids	Hydrocortisone suppository	30 mg (one suppository) twice daily
		Hydrocortisone enema	100 mg (one 60 mL unit) twice daily
	Oral 5-aminosalycylic acid (5-ASA) derivatives	Sulfasalazine	4 to 6 grams per day in four divided doses
		Mesalamine (Pentasa, Asacol)	2 to 4 grams daily in two to four divided doses
		Olsalazine capsule	2 to 3 grams daily in two to four divided doses
		Balsalazide capsule	2.25 to 6.75 grams daily in three divided doses
	Glucocorticoids	Prednisone or oral prednisolone	40 to 60 mg once each morning or in two divided doses
		Intravenous prednisolone	30 mg IV every 12 hours
		Methylprednisolone	16 to 20 mg IV every eight hours
		Hydrocortisone	100 mg IV every eight hours

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Role	Medication		Daily dose
intenance Remission	Topical (rectal) Mesalamine	Suppository	1 gram (1 suppository) at bedtime
		Retention enema	2 to 4 grams (30 to 60 mL) at bedtime
	Oral 5-aminosalycylic acid (5-ASA) derivatives	Sulfasalazine	4 to 6 grams per day in three or four divided doses
		Mesalamine (Pentasa, Asacol)	1.6 to 2.4 grams daily in one to three divided doses (depending upon preparation)
		Olsalazine capsule	1 gram daily in two divided doses
		Balsalazide capsule	2 to 6.75 grams daily in three divided doses

n's disease management:

smoking

ing remission:

) Glucocorticoids oral, topical or intravenous are generally used to induce remission. Budesonide is an alternative in a subgroup of patients

) Enteral feeding with an elemental diet may be used in addition to or instead of other measures to induce remission, particularly if there is concern regarding the side-effects of steroids (for example in young children)

) 5-ASA drugs (e.g. mesalazine) are used second-line to glucocorticoids but are not as effective

) Azathioprine or mercaptopurine may be used as an add-on assess□medication to induce remission but is not as monotherapy thiopurinemethyltransferase (TPMT) activity before offering azathioprine or Methotrexate is an alternative to azathioprine□mercaptopurine

) Infliximab is useful in refractory disease and fistulatingCrohn's. Patients typically continue on azathioprine or methotrexate. (Used also in rheumatoid arthritis)

) Metronidazole is often used for isolated peri-anal disease

Maintaining remission:

1) Stopping smoking Is a priority (remember: smoking makes crohn's worse, bu may help ulcerative colitis)

2) Azathioprine or mercaptopurine is used first-line to maintain remission

3) Methotrexate is used second-line

4) 5-ASA drugs (e.g. mesalazine) should be considered if a patient has ha previous surgery

Surgery:

Around 80% of patients with Crohn's disease will eventually have surgery

ADA CRITERIA FOR DIAGNOSING DIABETES

1. HbA1C ≥6.5 percent OR
2. FPG ≥126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least h. OR
3. Two-hour plasma glucose ≥200 mg/dL (11.1 mmol/L) during an OGTT. The te should be performed as described by the World Health Organization, using a glucos load containing the equivalent of 75 g anhydrous glucose dissolved in water. OR
4. In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, random plasma glucose ≥200 mg/dL (11.1 mmol/L).

Note: in the absence of hyperglycemic symptoms test should be repeated to confir diagnosis.

POST-EXPOSURE PROPHYLAXIS

Hepatitis A:

Human Normal Immunoglobulin (HNIG) or

hepatitis A vaccine may be used depending on the clinical situation

Hepatitis B:

1) HBsAg positive source: If the person exposed is a known responder to HB vaccine then; a booster dose should be given. If they are in the process of bein vaccinated or are a non-responder then; hepatitis B immune globulin (HBIG) an the vaccine should be given

2) Unknown source: for known responders the green book advises considering a booster dose of HBV vaccine. For known non-responders HBIG + vaccine should be given. Whilst those in the process of being vaccinated should have an accelerated course of HBV vaccine

Hepatitis C:

Monthly PCR, if seroconversion then interferon +/- ribavirin

HIV:

1) A combination of oral antiretrovirals (e.g. Tenofovir, emtricitabine, lopinavir and ritonavir) as soon as possible (i.e. within 1-2 hours, but may be started up to 72 hours following exposure) for 4 weeks
2) Serological testing at 12 weeks following completion of post-exposure prophylaxis
3) Reduces risk of transmission by 80%

Varicella zoster:

VZIG for IgG negative pregnant women/immunosuppressed

Estimates of transmission risk for single needle stick injury:

Hepatitis B: 20-30%

Hepatitis C: 0.5-2%

HIV: 0.3%

POOR PROGNOSTIC FACTORS

Acute myeloid leukaemia:

1) > 60 years
2) > 20% blasts after first course of chemotherapy.
3) Cytogenetics: deletions of chromosome 5 or 7

Acute lymphoblastic leukaemia (ALL):

Poor prognostic factors:

- FAB L3 type
- T or B cell surface markers
- Philadelphia translocation, t(9;22)
- age < 2 years or > 10 years
- non-Caucasian
- male sex
- CNS involvement

- high initial WBC (e.g. > 100 * 109/l)

Chronic lymphocytic leukaemia (CLL):

Poor prognostic factors: (median survival 3-5 years)

- Male sex
- Age > 70 years
- Lymphocyte count > 50
- Prolymphocytes comprising > 10% of blood lymphocytes
- Lymphocyte doubling time < 12 months
- Raised LDH 7) CD38 expression positive

Hodgkin's lymphoma:

Poor prognostic factors are

- Age > 45 years
- Male
- Stage iv disease
- Hemoglobin < 10.5 g/dl
- White blood count > 15,000
- Lymphocyte count < 600 or < 8% 7) Albumin < 40 g/l

Multiple Myeloma:

Poor prognostic factors include

- Raised B2-microglobulin
- Low albumin

Guillain-Barré syndrome:

Poor prognostic factors include:

- Older age
- Rapid onset (less than seven days) prior to presentation
- Severe muscle weakness on admission
- Need for ventilatory support
- An average distal motor response amplitude reduction to <20 percent of normal
- Preceding diarrheal illness

heumatoid arthritis:

- rheumatoid factor positive
- anti-CCP antibodies
- HLA DR4
- poor functional status at presentation
- X-ray: early erosions (e.g. after < 2 years)
- extra articular features e.g. nodules
- insidious onset

ypertrophic obstructive cardiomyopathy (HOCM):

- Syncope
- Family history of sudden death
- Young age at presentation
- Non-sustained ventricular tachycardia on 24 or 48-hour holter monitoring
- Abnormal blood pressure changes on exercise
- An increased septal wall thickness